EFFICACY OF LOW LEVEL LASER THERAPY IN ULCER FOOT HEALING IN TYPE 2 DIABETIC PATIENTS

Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery

(Branch I)

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

Chennai

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CERTIFICATE

Certified that this is the bonafide dissertation done by Dr.R.SENTHIL KUMAR and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai.

Date : *Unit Chief*

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DECLARATION

I solemnly declare that the dissertation titled **"EFFICACY OF LOW LEVELLASER THERAPY IN ULCER FOOT HEALING IN TYPE 2 DIABETIC PATIENTS "** was done by me from 2007 onwards under the guidance and supervision of **Associate Professor Dr. R.KATTABOMMAN & Professor Dr.N.JAYARAMACHANDRAN (Retd).**

This dissertation is submitted to the **Tamilnadu Dr. MGR Medical University** towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Place : **Dr. R.SENTHIL KUMAR**

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Course : M.S. General Surgery

Period of Study : 2007 - 2010

College : Coimbatore Medical College

Dissertation Topic : Efficacy of low-level laser therapy in ulcer foot healing in type-2 diabetic patients.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 13.02.08

Secretary
Ethics Committee

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Place : Coimbatore ***Dr.R.Senthil Kumar***

LIST OF ABBREVATIONS

Hb	: Haemoglobin
mm	: Millimeter
Vs	: Versus
min	: Minutes
PR	: Pulse Rate
BP	: Blood Pressure
DBP	: Diastolic Blood Pressure
SBP	: Systolic Blood Pressure
DM	: Diabetes Mellitus
L.L.L.T	: Low level laser therapy
US – FDA	: United States Food and Drugs Administration
Laser	: Light Amplification by Stimulated of Emission of Radiation

ABSTRACT

Efficacy of low level laser therapy in diabetic wound healing diabetic wound healing dynamics

A Randomized Control Trial

Research question : *Is low level laser therapy given once in three days for 20 days (7 sessions) cluster probe at 2.5 m w at a dose of 8 - 10 J/cm2, CO_2 Laser along with conventional therapy compared to conventional therapy alone effective in reducing the mean percentage in area reduction in diabetic foot ulcer participants treated in coimbatore medical college.*

Objective:

To determine mean percentage reduction of wound area in either group

Study design:

A randomized control study from oct 2007 – sep 2009

Setting:

Coimbatore Medical College Hospital, Coimbatore

Sample size:

Forty participants (20 in each group) with type II diabetes with foot ulcers who fulfilled the eligibility criteria participated in the study.

Randomization procedure:

Randomization was done using a computer generated randomization chart.

Method :

After the initial wound measurement on day 1, study group received L.L.L.T with conventional therapy (saline or betadine dressings) for a period of 20 days while the control group received only conventional therapy (Saline or Betadine dressings). Wound was reassessed for area reduction on day 20.

Out come measures:

Percentage of area of wound reduction were measured using unpaired student T Test

Results:

Participants in the study group had better mean percentage wound contraction 41.17% as compared to the control group 15.55% ($p < 0.001$ $Z = 7.08$).

Conclusion :

Low level laser therapy can be used as an adjunct to conventional therapy for the healing of diabetic foot ulcers.

Key words: diabetic foot, low level laser therapy

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INTRODUCTION

The incidence of diabetes and its complications are on a rise, the risk of lower extremity amputations is 15 fold higher in diabetics as compared to non – diabetics. essential to mention here that chronic diabetic foot ulcer is the leading cause of amputations in these patients, also that 15% of all diabetics develop diabetic ulcer and the most commonest site being the foot. although the fundamental pathophysiologic factors leading to diabetic ulcer remain incompletely understood. The triad of neuropathy. Ischemia and infections commonly is considered the most important. These diabetic ulcers are known to be resistant to conventional treatment and may herald severe complications if not treated wisely.

Research findings to date based upon animal, human and most notably invitro cellular studies have certainly established the fact, that low level laser therapy can play a major, adverse effect free useful role in healing chronic diabetic ulcers resistant to the conventional mode of treatment.

Reports of low laser therapy applied to soft tissues in vitro and in vivo suggest stimulations of specific healing processes in healing wounds. A common feature of these studies are that while low does of laser therapy are simulative the higher doses are suppressive.

Major changes seen in the wounds treated with low level laser therapy include increased granulation tissue, increased granulation tissue, increased fibroblast proliferation, collagen synthesis, epithelializaion and enhanced neovascularizatin. In humans, anecdotal clinical observations of small case studies have suggested that low level laser therapy stimulates the process of healing. A criticism of many in vivo experimental designs to examine the efficacy of low level laser therapy has been lack of control, port experimental design, inequality among the control and the study groups

and inadequate specification of treatment settings, because of very few studies in humans and previous studies with ill defined controls and poorly defined and documented parameters we felt the need to undertake this study.60 various studies have been performed to improve the understanding of low level laser therapy effects at cellular level. Early research suggested laser mediated improvements in wound healing recent cold laser studies have further focused on laser impact on cell growth. Because fibroblasts play a crucial role in wound healing, most of the studies published in the in the LLT literature[5] have examined the effects LLT on fibroblast cell growth, locomotion and production of collagen, in early 90;s a direct and massive transformation of cultured fibroblasts into my fibroblasts was observed as early as 24 hours after laser treatment; this in vitro induction may be analogous to that of in vivo. Thus induction of a phenotype with contractile properties may have clinical significance in the acceleration of wound healing.

More recently studies done on effects of LLLT irradiation on cell[15] proliferation of he la cells also showed encouraging results and most of the effects observed were at molecular level. Growth curves of all cell population were increased regardless of cell irradiation, though most encouraging results were encountered after 24 to 96 hrs after irradiation. Most studies were done in a pulsed frequency of 5 kHz and energy densities between 4 – 12 J/ cm2[4]

Most of the research work done with respect to LLLT has been with recalcitrant ulcers, venous ulcers[3], pressure sores radiation ulcers. There are a few case reports and some poor quality studies on diabetic foot ulcers[15]. Indirect evidence in a study suggest improvement in skin circulation in patients with diabetic microangiopathy due to athermic effects of laser irradiation. Another study used topical hyperbaric oxygen and LLLT in treatment of chronic diabetic foot ulcers with positive outcome and stated that LLLT may be safe and simple treatment in such patient.

Hence to keep the controls and the study group alike as much as possible a triple blind sham controlled study was done to assess the efficacy of LLLT on incised wounds in experimental human model and were observed for wound contraction, colour change, and luminance, the study supported the fact that LLLT improves healing rates in superficial incised wounds, but again these subjects were not diabetics, and the efficacy in diabetic patients still remains obscure.

In view of inadequate studies regarding the efficacy of LLLT in diabetic wound healing dynamics we undertook this study to know whether low level laser therapy given daily with clustered probe at 2.5mW at 5KHz frequency, 10,600 nm wavelength, 2 – 5 min at a dose of 8 - 10 J/cm2 along with conventional treatment compared to conventional therapy alone, is effective in reducing the mean percentage in area reduction, In patients with diabetic foot ulcers more than 3 weeks old admitted in cmch under surgery department.

AIMS AND OBJECTIVE

- *To test the efficacy of low level carbon - dioxide [CO_2] laser therapy with conventional therapy vs conventional therapy alone in Type 2 diabetic foot ulcer patients treated in Coimbatore Medical College Hospital Coimbatore, between October 2007 and September 2009.*

- *To determine mean percentage of wound area reduction in either group.*

REVIEW OF LITERATURE

HEALING

Definition

"Body replacement of destroyed tissues by the living tissue" or "integrated series of cellular & biochemical events which restores the functional integrity & regains the strength of injured tissue"

Phases of Healing

Wound healing & repair are complex processes that involves dynamic series of event [Reddy G.K.2001]

- **Coagulation**
- **Inflammation**
- **Fibroplasias, angiogenesis, proliferation & granulation tissue formation**
- **Epitheliazation**
- **Collagen synthesis**
- **Wound contraction / tissue remodeling / scar maturation**

HISTORICAL BACKGROUND OF LASER

The word LASER is an acronym for Light Amplification by Stimulated Emission of Radiation

According to the European standard IEC 601, the definition of laser is "any device which can be made to produce or amplify electromagnetic radiation in the wavelength range from 180 nm to 1 mm, primarily by the process of controlled stimulated emission".

TABLE No 3.1: HISTORICAL BACKGROUND OF THERAPEUTIC LASER

Scientist	Year	Contribution Made in Development of Laser
Albert Einstein	1916	Principle on which lasers are based were postulated. Credit for the development of laser theory is generally given to Elbert Einstein. In his theory "Zur Quantum Theories Der Strahlung", published in 1916, he first used the name "stimulated emission".
Mester Et al	1985	Non invasive lasers were introduced into medicine in 1980's, since then, have gained wide application in many areas of health care.
Lam TA Et al	1986	Although the beneficial effects of laser photostimulation are now generally accepted, the mechanism by which laser facilitates the wound healing & tissue repair, is yet to be clearly understood
Lysons Rf	1987	
Kert Et al	1989	The non invasive low power lasers up to 500 mw have been reported to have stimulatory, anti – inflammatory & analgesic effects
Meller mm	1990	The beam of laser energy is coherent, polarized, focused & monochromatic.
Schindl A et al	1998 – 2000	Low energy lasers are effective as analgesic & accelerates the healing of injured tissues.

COLD LASER

Lasers used in field of medicine are known as cold lasers. The power levels of light are greatly amplified by the emission of radiation from stimulation of specific substances. Every substance radiates emission in varying wavelengths and frequencies. Helium, neon, cobalt, and carbon dioxide are examples of substances that, when irradiated, have application in commercial, medical or engineering processes.

MECHANICS

To obtain the laser, a tube filled with a gaseous mixture of helium and neon is stimulated electrically to emission levels. Within the highly reflective, polished walls of the tube, the molecules reverberate and carom off the walls in a highly agitated state, building energy as they do so. When a critical level is reached, the flow of energy literally "bursts" through the semi silvered (similar to one-way mirror) front end of the tube and is channeled along an optic fiber to the beam applicator or probe for clinical applications.

LASER PHYSICS

Intensity

One might naturally assume that the laser generates tremendous power; however, the type of laser used by physical therapist has the intensity of only one m W-less than the power of a 60-W commonly used light bulb! The commercial and industrial (i.e. hot lasers) range in the thousands and millions of watts and are used for cutting, drilling and destructive applications. The tool used by physical therapists, a cold laser, is used primarily for healing and other nondestructive purposes.

PHYSICAL CHARACTERISTICS

There are three characteristics of laser light that clearly differentiate it from ordinary light.

I-MONOCHROMATICITY

Ordinary light is comprised of a conglomeration of many wave lengths, commonly known as ROYGBIV, or the visible spectrum of Red, Orange, Yellow, Green, Blue, Indigo, and Violet, all merging to produce "white" light. Laser light, however, consists of one wave length only which is 6,328 A units. Because this wave length falls within the R section of the visible spectrum (3,900 to 7,700 A units), the laser light of 6,328 A units is a brilliant red color.

2-COHERENCE

Because the wave lengths of ordinary light are so variable and do not "match" in wave forms, frequencies, or shapes, there is much scrambling of wave forms, cancellation and reinforcements of individual waves, and interference in the production of energy in general; this factor minimizes the power of ordinary light as an energy source. The identical wave lengths and forms, that comprise laser light, lead to great amplification since the "waves and troughs" of the radiation are reinforced. Because they are parallel and in line with each other, they are termed coherent.

3-NON-DIVERGENCE

The laser beams unique in the absolute "straightness" of the directed radiation. Ordinary light shines in all directions (e.g. consider a light bulb radiating in all directions). The sun is another example of omni directional radiation. The laser, on the other hand, shines in only one direction, not unlike a flashlight, although its beam is far more concentrated and narrowed. The divergence of a laser beamed to the surface of the Moon and Earth showed a deflection of just a few meters after a journey of more than 260,000 miles.

4. OUTPUT POWER = STRENGTH (W/Mw)

- **Power is measured in watts (W). the strength or power output of a laser is thus measured in watts or milliwatts (mW=A thousand of watt)**
- **Higher output power means higher power density, which is often desirable**
- **In addition, a higher output power means that a certain desired dose (input energy. Measured in joules per cm2 of skin) is more quickly reached because energy is the same as power multiplied by time.**

ENERGY :

Joules	**=**	**Power (mW) X Time (Sec)**
Eg Joules	**=**	**2.5mW X4sec**
	=	**10J/cm2**

PHYSIOLOGY

1. WOUND HEALING

Ordinary light does not penetrate the skin or underlying tissues. Only the retina can absorb the visible spectrum. As discussed previously, infrared is absorbed at the 3-mm level and ultraviolet at the 1-mm level. Research has indicated that the 6,328 A unit wave length of the cold laser may stimulate intracellular structures and functions. One of the prime applications of laser light in physical therapy is that of wound healing.

2. ANALGESIC EFFECTS

In pain control, the penetration of laser light is often compared with an acupuncture needle. Therefore, when the laser is used for analgesic purposes, the beam is generally directed at acupuncture points, trigger points, and nerve roots, as is done with acupuncture.

3. PENETRATION

Direct penetration of the 6,328 A unit HeNe cold laser at I m W said to be approximately 0.8mm; indirect penetration after reflection, dispersion, reflection, and partial absorption is 10 to 12mm.

4. ABSORPTION

Absorption is apparently dependent on the resonance of the tissues or on the subject's water content. Many of the penetration characteristics and limitation factors are determined by fluids (e.g. water, blood).

PHYSICAL EFFFECTS

1. HEATING

A mild but reversible heat is produced with the cold laser. The tissues revert back to the pre-lasting temperatures immediately following radiation. Although the thermodynamics involved do not produce a therapeutic level, cell wall permeability has been shown to be a favorable result of heat and may play a role in the reaction of the laser on the cell wall.

2. DEHYDRATION

Loss of water following radiation is another reversible process. Such loss may be attributed to the minor heating and/or the transfer of fluids to distant sites. Apparently, it does not play a major role in the laser's effectiveness.

3. COAGULATION OF PROTEINS, THERMOLYSIS, AND EVAPORATION

Coagulation of proteins, Thermolysis, and evaporation are irreversible and should not occur with the dosages and techniques used by physical therapists. Coagulation is a permanent process, comparable to that of an egg frying in a skillet. Thermolysis or "melting from heat" is also a lasting state. Evaporation, of course, involves the transformation of

liquids to the gaseous state and is not easily reversed. With competent clinicians and sophisticated equipment, none of these conditions should be produced in routine administration of the cold laser. With higher power levels, one must be cautious and aware of the above factors.

INDICATIONS

Cold laser is indicated for treating:

- **Open lesions**
- **Decubitus ulcers**
- **Diabetic ulcers**
- **Lacerations**
- **Incisions**
- **Burns**
- **Chronic and acute pain especially those of musculoskeletal origin. For example, arthritis, sprains tendonitis, contusions, lumbago, neuralgia, neuritis.**
- **Restricted joint ranges of motion**

CONTRINDICATIONS

- **Do not radiate the eye directly.**
- **Whether is suspected due to its mobilizing effect on steroids in the human system.**
- **Do not use the laser with patients who are naturally photosensitive or who are photosensitized by medication.**
- **Patients with pacemakers (Since the electronic circuits of the laser system could interfere with the operation of the pacemaker).**
- **Patients with left ventricular insufficiency under treatment for poor peripheral circulation (since laser treatment might produce a dangerous overloading of the central venous system).**
- **Patients with recent heart attacks.**

RISK OF EYE INJURY

Even in the infancy of laser technology there was an awareness of the increased risk of eye damage compared to conventional light sources, hence, new rules introduced to handle this hazard, lasers were divided into five categories (class 1,2,3A,3B and 4) according to their potential to damage the eye.

TABLE NO 3.2 : RISK OF EYE INJURY

Class	Safety with Regard to Eye Damage
Classes 1-3A	Are considered safe
Class 3B	Involves a certain risk
Class 4	Definite risk Comprises strong industrial and surgical lasers capable of burning and cutting

Note that this classification has nothing to do with the medical use, efficiency or quality of lasers but refers solely to the possible risk of eye injury.

THE FOLLOWING AREAS SHOULD NEVER BE IRRADIATED:

- **Eyes and pregnant uterus as mentioned above.**
- **Thyroid glands (due to the possibility of increased hormone secretion).**
- **Male external genitalia (due to the possibility of interaction with the cells of the germinal line or the endocrine portion of the testicle**
- **Skin if there is cutaneous or subcutaneous bacterial infection.**
- **Growing cartilage in children.**

PRECAUTIONS

Poor results may ensure in patients:

- **Of extreme age**
- **Under heavy medication**
- **With thick eschar**
- **With considerably scar tissue**
- **With extremely dry skin**
- **With active infection**

A touch of moisture on the tip of probe or the target skin may enhance the electrical contact needed for efficient point searching. Perspiration or other skin moisture will naturally give false read-outs; adjustments must be made in such circumstances. Dry the patient's skin to lasing and/or select higher readings on the LED for targeting.

WOUND HEALING & REPAIR

PRINCIPLE OF ARNDT – SCHULTZ

- **If the quantity of energy is too small to stimulate the absorbing tissues, NO significant reaction will take place.**
- **If the quantity of energy absorbed per unit of time is adequate to stimulate, the absorbing tissue will perform its normal function.**
- **It the quantity for energy is too great, per unit time, the absorbing tissue will be disrupted and cannot perform its normal function.**
- **Local tissue temperatures SHOULD NOT be elevated above 45oc that tissue destruction is likely to occur.**
- **Therefore proper selection of laser dose is necessary to prevent photo – inhibitory effects (as it works on the principle of ARNDT – SCHULTZ).**

- **Effects of LLLT are photochemical not thermal and the responses of cells occurs due to changes in photo acceptor molecules (also known as chromophores molecules which are able to absorb photonic energy) such as porphyrin.**

- **The exact mechanism of action of LLLT is not completely understood. However, it is known that during laser irradiation cells absorb photonic energy that is incorporated into chromophores, which in turn, stimulates cellular metabolism**

- **The chromophores is able to transfer the absorbed energy to other molecules and thus cause chemical reactions in surrounding tissue.**

DOSAGE

Actual dosages with the cold laser depend on the power factor, duration of radiation, and tissue resonance.

WOUND HEALING

In most wound healing applications, the prescribed dosage is 10,600 nm wavelength, 2-10 sec/cm2 of open lesion. This may require several minutes of hand – held direction of the beam over the surface of the wound so that each square centimeter is exposed for the same 2-10 seconds. The probe tip is held approximately 2 to 3 mm from the surface to obtain a "Disc" of laser light about 1 cm in diameter on the surface of the wound. The non – divergent nature of the laser is modified by a lens – like spreading of the beam for therapeutic purposes.

LASERS IN MEDICINE AND SURGERY

In the table below, the names, wavelengths, working modes, and uses of the most common laser systems used in medicine are listed.

TABLE NO 3.3 : CLASSIFICATION OF THERAPEUTIC LASERS

LASER NAME	WAVELENGTH (NM)	PULSED OR CONTINUOUS	MEDICAL USE
Crystalline laser Medium			
KTP / 532	532 nm	p/c	Leg Vein Treatment
Ruby	694	P	Tattoo & Hair Removal
Alexandrite	755	P	Bone Cut, Hair Removal
ND : YAG	1064	P	Coagulation of Tumors
HO : YAG	2130	P	Surgery, Root Canal, lithotripsy
ER : YAG	2940	P	Dental Drill, Laser Peeling
Ti: Sapphiere	Tunable	P	Two Photon PDT
Semiconductor Lasers			
InGaAIP	630-700	C	Biostimulation
Liquid laser medium			
Dye Laser	Tunable	P/C	Kidney Stones
Gas Lasers			
Excimer	193, 248, 308	P	Eye, Vascular Surgery
Argon	350-514	C	Dermatology, Retinopathy
Copper	578	P/C	Dermatology
HeNe	633,3390	C	Biostimulation
Co2	10600	P/C	Dematology, Surgery

THE PRODUCTION OF LASER RADIATION BY CARBON DIOXIDE LASER

The carbon dioxide laser (CO_2 laser) was one of the earliest gas lasers to be developed (invented by Kumar Patel of Bell Labs in 1964[1]), and is still one of the most useful. Carbon dioxide lasers are the highest-power continuous wave lasers that are currently available. They are also quite efficient: the ratio of output power to pump power can be as large as 20%.

The CO_2 laser produces a beam of infrared light with the principal wavelength bands centering around 9.4 and 10.6 micrometers.

AMPLIFICATION

The active laser medium (laser gain/amplification medium) is a gas discharge which is air cooled (water cooled in higher power applications). The filling gas within the discharge tube consists primarily of:

- **Carbon dioxide (CO_2) (around 10–20 %)**
- **Nitrogen (N_2) (around 10–20%)**
- **Hydrogen (H_2) and/or xenon (Xe) (a few percent; usually only used in a sealed tube.)**
- **Helium (He) (The remainder of the gas mixture)**

The specific proportions vary according to the particular laser.

The population inversion in the laser is achieved by the following sequence:

1. **Electron impact excites vibrational motion of the nitrogen. Because nitrogen is a homonuclear molecule, it cannot lose this energy by photon emission, and its excited vibrational levels are therefore metastable and live for a long time.**

2. **Collisional energy transfer between the nitrogen and the carbon dioxide molecule causes vibrational excitation of the carbon dioxide, with sufficient efficiency to lead to the desired population inversion necessary for laser operation.**

3. **The nitrogen molecules are left in a lower excited state. Their transition to ground state takes place by collision with cold helium atoms. The resulting hot helium atoms must be cooled in order to sustain the ability to produce a population inversion in the carbon dioxide molecules. In sealed lasers, this takes place as the helium atoms strike the walls of the container. In flow-through lasers, a continuous stream of CO_2 and nitrogen is excited by the plasma discharge and the hot gas mixture is exhausted from the resonator by pumps.**

The CO_2 laser can be constructed to have CW powers between milliwatts (mW) and hundreds of kilowatts (kW).[2] It is also very easy to actively Q-switch a CO_2 laser by means of a rotating mirror or an electro-optic switch, giving rise to Q-switched peak powers up to gigawatts (GW) of peak power[3].

DIABETES MELLITUS

Definition

"Diabetes mellitus is characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both".

Depending on the etiology of the DM, factors contributing to hyperglycemia may include:

- **Reduced insulin secretion**
- **Decreased glucose utilization**
- **Increased glucose production**

Or

"Level of glycaemia at which diabetes specific complications occur rather than on deviations from population based mean"

Epidemiology of the diabetic foot syndrome

- **A quarter of the diabetic population is at increased risk of foot injuries as a result of the presence of diabetic neuropathy or an arterial circulatory disorder. Every year 3 to 7% of diabetics suffer a foot lesion for the first time.**

- **Foot ulcers occur in approximately 15% of people with diabetes which accounts for 25% of all hospital admissions with the hospital stay being 60% longer than the stay for other causes and the risk of amputation is 15 to 40 times greater in diabetics that in others.**

- **Diabetic foot ulcers account for more than 50% of non traumatic amputations and are associated with high rates of mortality. Re – amputation and contra lateral limb amputation.**

- **India has 30 million diabetics at present and in the year 2025 India is predicted to have 57 million diabetics.**

INCIDENCE IN INDIA

Foot ulcer	**:**	**1-4%**
Toe amputation	**:**	**2.6%**
Below knee amputation	**:**	**1.6%**
Prevalence of diabetic foot in India	**:**	**5.3 – 10.5%**

SOCIO – ECONOMIC IMPACT OF THE DIABETIC FOOT SYNDROME

- **Overall, the costs generated by diabetes are about three times as high as those produced by non – diabetics. Foot complications constitute a major proportion of these.**

- **With primary healing, about 30% of the total cost derives from hospitalization, but where amputation is required this figure is 65% to 80% the average healing duration for diabetic foot lesions is about four months.**

- **Ten percent of all lesions persist for more than one year, which incurs further costs for outpatient care. Fifteen percent of all foot ulcers in diabetics do not heal before the patient's death**

- **Most important cost item is namely the "cost" to the patient themselves in terms of the emotional trauma suffered and the loss of quality of life and independence**

CLASSIFICATION

Type 1

Type pathology

1A : Autoimmune beta cell destruction ⟶ Insulin deficiency

1B : Develop insulin deficiency by unknown mechanism causing destructive process of beta cells
Lack immunologic markers

Type II

It is a heterogeneous group of disorders characterized by

- **Impaired insulin secretion**
- **Variable degree of insulin resistance**
- **Increased glucose production**

Distinct genetic & metabolic defects in insulin action & or secretion give rise to the common phenotype of hyperglycemia in type – 2 DM

Type – 2 DM is preceded by a period of abnormal glucose homeostasis classified as

- **Impaired fasting glucose (IFG)**
- **Impaired glucose tolerance (IGT)**

Diagnosis

The National Diabetic Date Group & World Health Organization have issued a diagnostic criteria for DM-2 based on the following facts:

- **RBS >200 mgs / dl or > 11.1 m mol / l with symptoms of DM (polyuria, polydipsia, weight loss)**

- **FBS > 126 mgs / dl or > 7.0 m mol / L**

- **2 hr plasma glucose (during oral GTT) > 200 mgs / dl or >11.1 m mol / L (Not recommended a part of routine screening)**

TABLE NO 3.4 : DIAGNOSIS OF DIABETES MELLITUS

Terms	Definition
Random blood glucose (RBS)	Blood glucose levels with out regard to time since last meal
Fasting blood glucose (FBS)	Blood glucose levels when there is no caloric intake for past 8 hrs
2 Hr plasma glucose (During Oral GTT)	The test should be performed using a glucose load containing the equivalent of 75 gms anhydrous glucose dissolved in water

Type 2 DM

Etio – pathogenesis

- Insulin resistance
- Abnormal insulin secretion

Most studies support the view that insulin resistance precedes insulin secretary defects and that diabetes develops only if insulin secretion becomes inadequate.

Genetic considerations

Type 2 DM has a strong genetic component

- The concordance of type 2 DM in identical twins is between 70 and 90%
- Individuals with a parent with type 2 DM have an increased risk of diabetes; if both parent have type 2 DM, the risk approaches 40%
- Insulin resistance, as demonstrated by reduced glucose utilization in skeletal muscle, is present in may non diabetic, first – degree relatives of individuals with type 2 DM

- **Major genes that predispose to this disorder have yet to be identified, but it is clear that the disease is polygenic and multifactor. The gene for the protease, calpain 10, is associated with type 2 DM in Hispanic and some other populations**

- **Various genetic loci contribute to susceptibility, and environmental factors (such as nutrition and physical activity) further modulate phenotypic expression of the disease. However, definition of the genetic susceptibility remains a challenge because the genetic defect in insulin secretion or action may not manifest itself unless an environmental event or another genetic defect, such as obesity, is superimposed.**

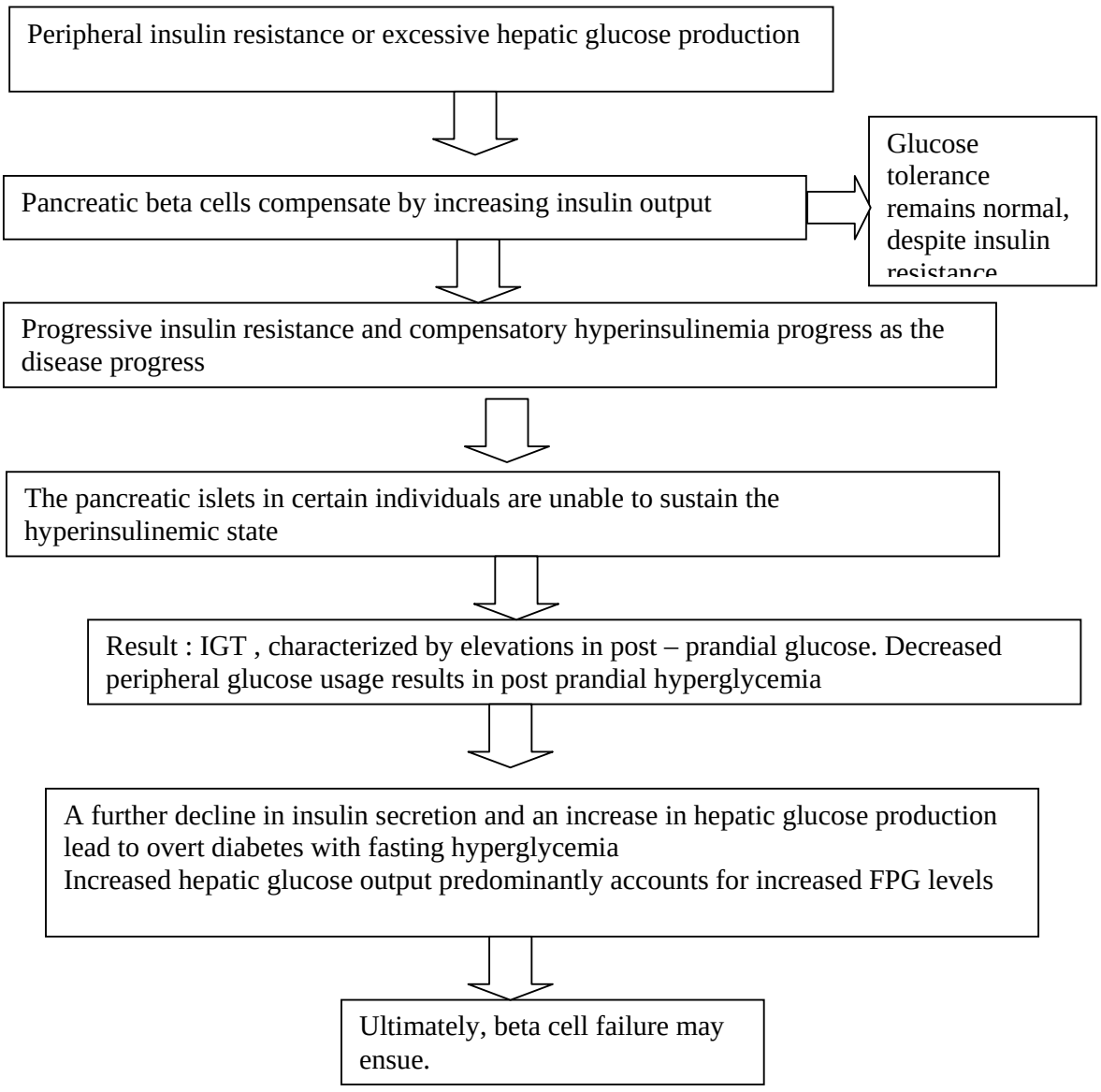
- **Mutations of molecules involved in insulin (e.g., the insulin receptor and enzymes involved in glucose homeostasis) account for a very small fraction of type 2 DM.**

Pathophysiology

Type 2 DM is characterized by three pathophysiologic abnormalities.

- **Impaired insulin secretion,**
- **Peripheral insulin resistance,**
- **Excessive hepatic glucose production**

Pathophysiology of type – II diabetes mellitus



Neuropathy and Diabetes Mellitus

- **The prevalence of diabetic neuropathy in patients with type 2 diabetes is 33 percent over all and more than 50% in patient over 60yeaer of age**
- **Diabetic neuropathy correlates with the duration of diabetes and glycemic control) type 2 DM.**
- **May manifest as**
    1. **Polyneuropathy**
    2. **Mono-neuropathy**
    3. **Automatic Neuropathy**
- **Both myelinated and unmyelinated nerve fibers are affected.**
- **Because the c/f of diabetic neuropathy are similar to those of other neuropathies, the diagnosis of diabetic neuropathy should be made only after other possible etiologies are excluded.**

Poly-neuropathy/Mono-neuropathy :

- **The most common form of diabetic neuropathy is distal symmetric polyneuropathy.**
- **It presents as**
    1. **Distal sensory loss – most frequent presentation**
    2. **Hyperesthesia**
    3. **Paresthesia**
    4. **Dysesthesia**
- **Symptoms includes a sensation of following, which begins in the feet & spreads proximally**
    1. **Numbness**
    2. **Tingling**
    3. **Sharpness**
    4. **Burning**

Any combination of these symptoms may develop as neuropathy progresses

- **Physical examination reveals**

    1. **Sensory loss**
    2. **Loss of ankle reflexes**
    3. **Abnormal position sense**

- **Pain typically involves lower extremities, is usually present at rest, and worsen at night.**

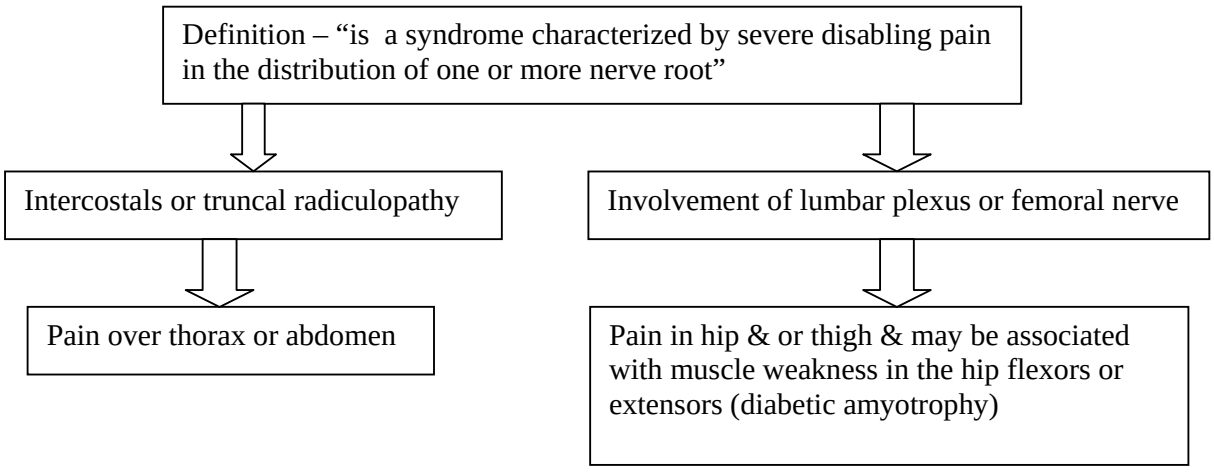
- **Both an acute (lasting <12 months) and a chronic form of painful diabetic neuropathy have been described.**

- **As diabetic neuropathy progresses, the pain subsides & eventually disappears, but a sensory deficit in the lower extremities persists.**

- **Neuropathic pain develops in some of these individuals, occasionally preceded by improvement in their glycemic control.**

Diabetic Neuropathy

If may be accompanied by – motor weakness



Treatment of Diabetic Neuropathy

- **Improved glycemic control should be pursued and will improve nerve conduction velocity, but the symptoms of diabetic neuropathy may not necessarily improve.**
- **Avoidance of neurotoxins (alcohol), supplementation with vitamins for possible deficiencies (B12, B6, folate).**
- **Symptomatic treatment**
- **Since pain of acute diabetic neuropathy may resolve over the first year, analgesics may be discontinued as progressive neuronal damage from DM occurs.**
- **Chronic, painful diabetic retinopathy is difficult to treat but may respond to**
    1. **Tricyclic antidepressants – amitryptline, desipramine, nortriptyline**
    2. **Gabapentin**
    3. **NSAIDs (avoid in renal dysfuntions)**
    4. **Others (mexlitne, phenytoin, carbamazepine, capsaicin cream)**
- **Referral to pain management center may be necessary.**

Lower extremity complications

- **Foot ulcers and infections are a major source of morbidity in individuals with DM.**
- **The reasons for the increased incidence of these disorders in DM involve the interaction of several pathogenic factors.**

- **Neuropathy**
- **Abnormal foot biomechanics**
- **Peripheral arterial disease**
- **Poor wound healing.**

NEUROPATHY

Neuropathy is present in over 80 percent of patients with foot ulcers

Peripheral Sensory Neuropathy

Interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, often without knowledge of the injury.

Motor and Sensory Neuropathy

Lead to abnormal foot muscle mechanics and to structural changes in the foot (hammer toe, claw toe deformity, prominent metatarsal head, charcot joint).

Autonomic Neuropathy

Results in anhidrosis and altered superficial blood flow in the foot, which promote drying of the skin and fissure formation.

Peripheral arterial disease and poor wound healing

Impede resolution of minor breaks in the skin, allowing them to enlarge and to become infected.

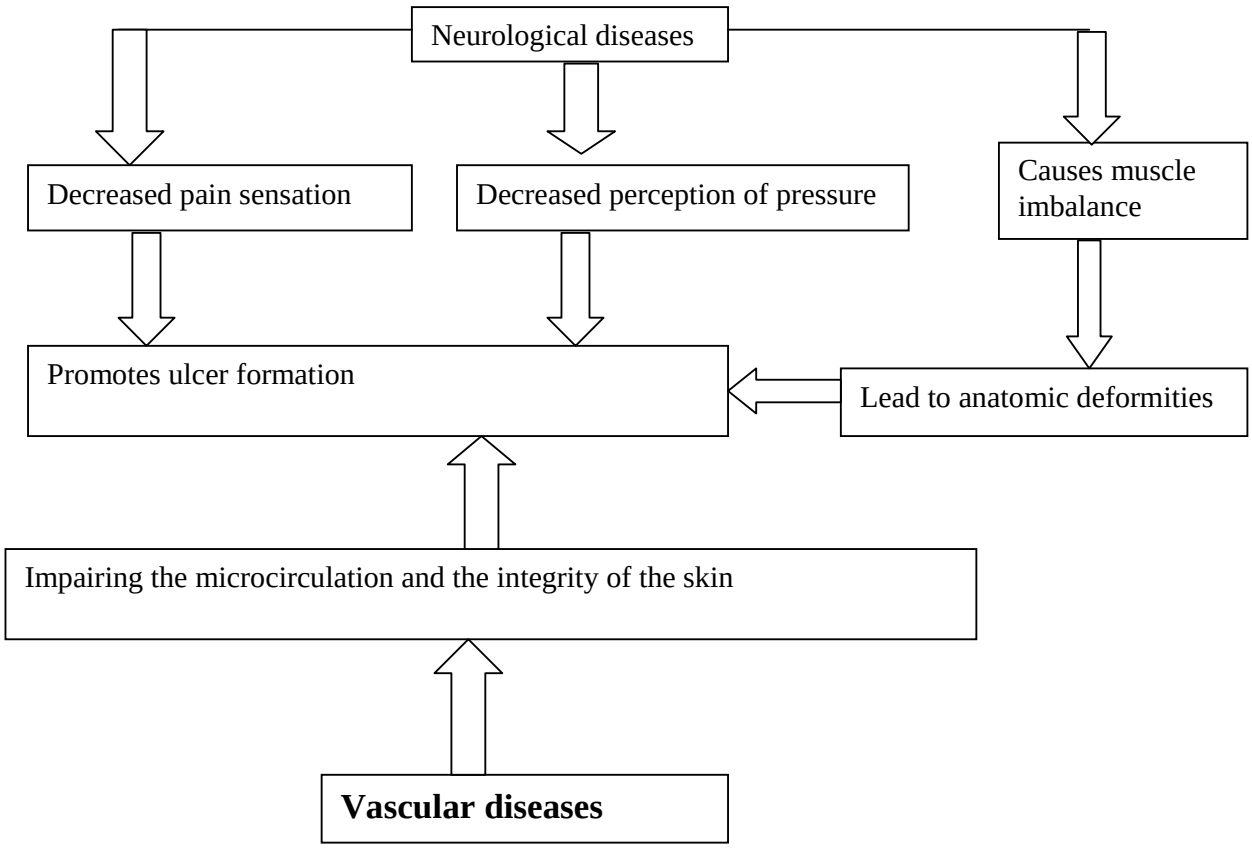
Disordered Proprioception

Causes abnormal weight bearing while walking and subsequent formation of callus or ulceration.

Approximately 15% of individuals with DM develop a foot ulcer, and a significant subset will ultimately undergo amputation (14 to 24%) risk with that ulcer or subsequent ulceration.

Foot problems are an important cause of morbidity in patients with diabetes mellitus

Pathogenesis of Diabetic Foot

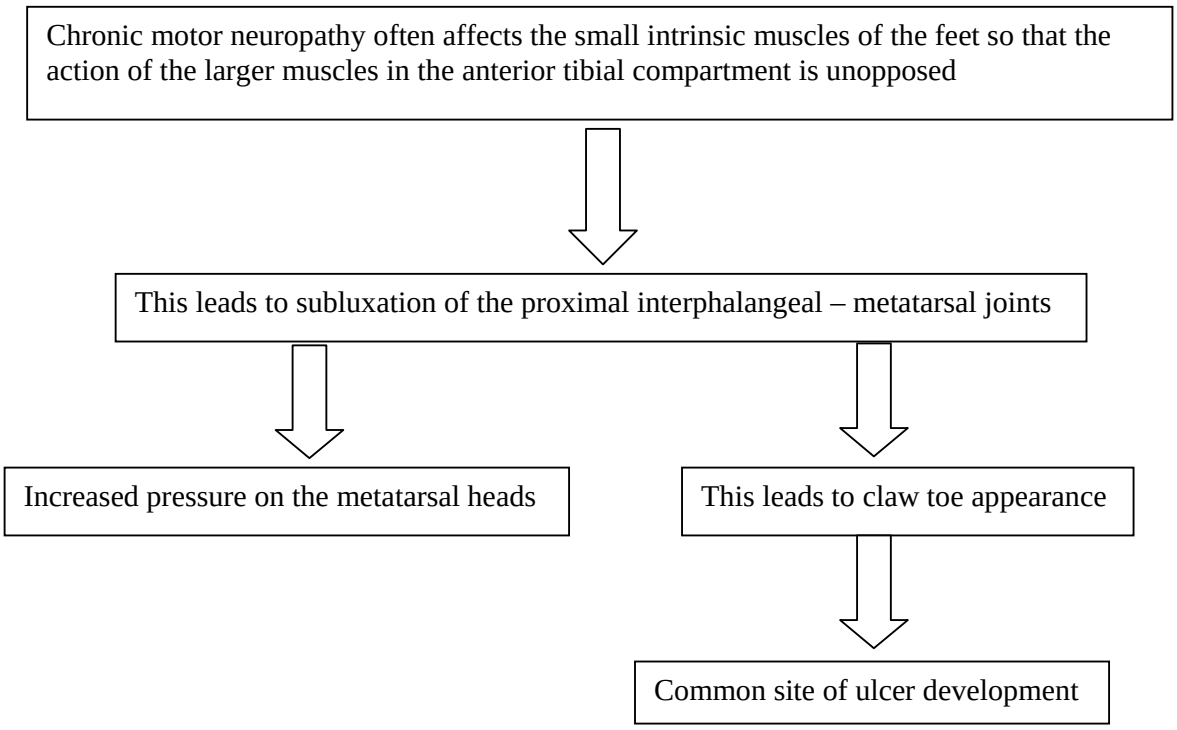


Physical signs resulting from diabetic neuropathy

The physical examination may reveal several abnormalities that result from neuropathy, such as

- **Claw toes**
- **Charcot arthropathy (also called diabetic neuropathic anthropopathy)**

Pathophysiology of Charcot Arthropathy



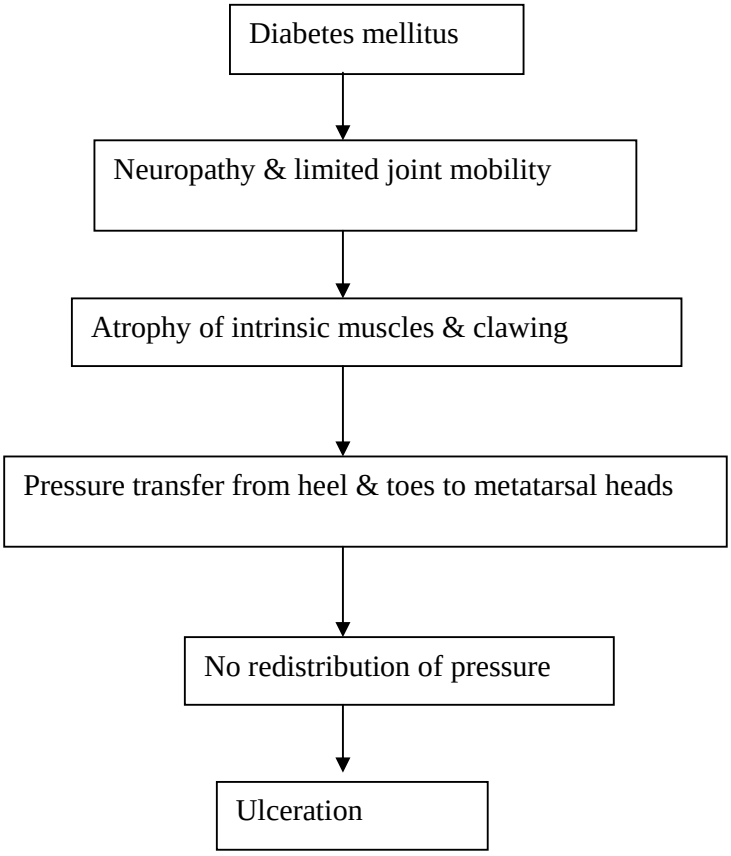
VASCULAR CHANGES IN DIABETES (Levin)

1. Atherosclerosis : chronic inflammatory process that can be converted into acute clinical events by plaque rupture (Berliner)

Development of atherosclerosis is accelerated in DM leading to increased morbidity and mortality. All the large vessels are involve in this process and clinical manifestations are apparent as a result of atherosclerotic narrowing and thrombosis of coronary, cerebral and leg vessels.

PATHOGENESIS OF DIABETIC ULCERS

Foot pressure abnormalities in diabetic foot



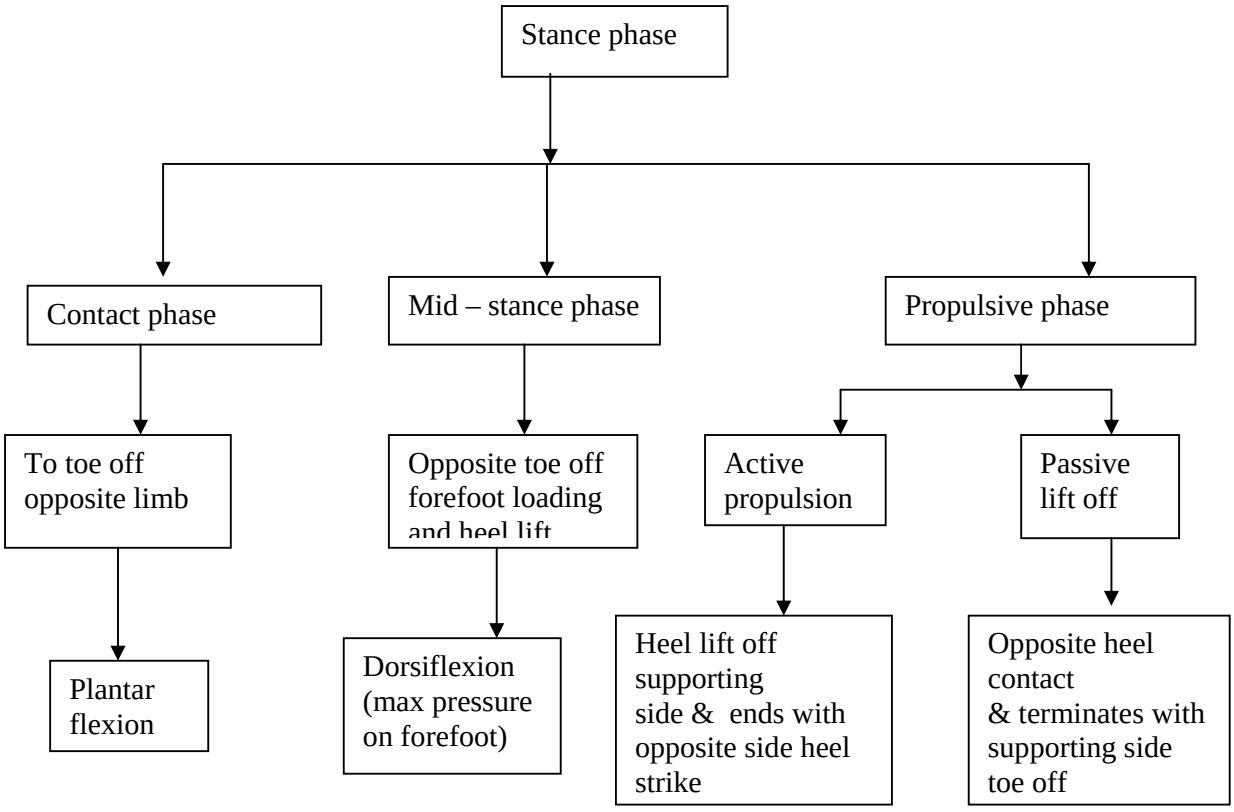
Predisposing Factors for Ulceration

1. **Limited joint mobility**
2. **Peripheral neuropathy**
3. **High plantar pressure**
4. **Vascular diseases**

Biomechanics of Diabetic Foot

Gait cycle:

1. **Stance phase**
2. **Swing phase**

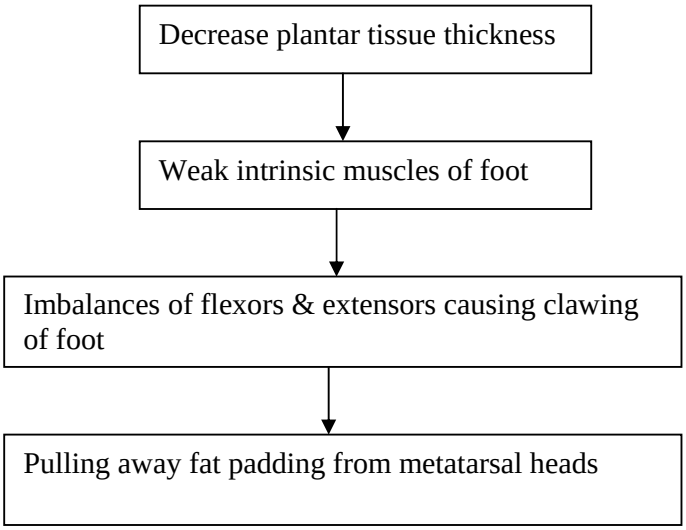


Changes in Foot Caused by Diabetes:

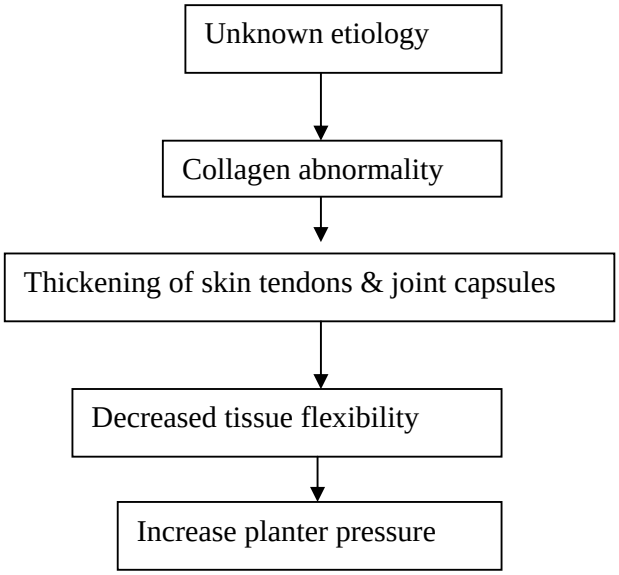
1. Peripheral neuropathy

A. **Dryness of skin**
B. **Callus formation**

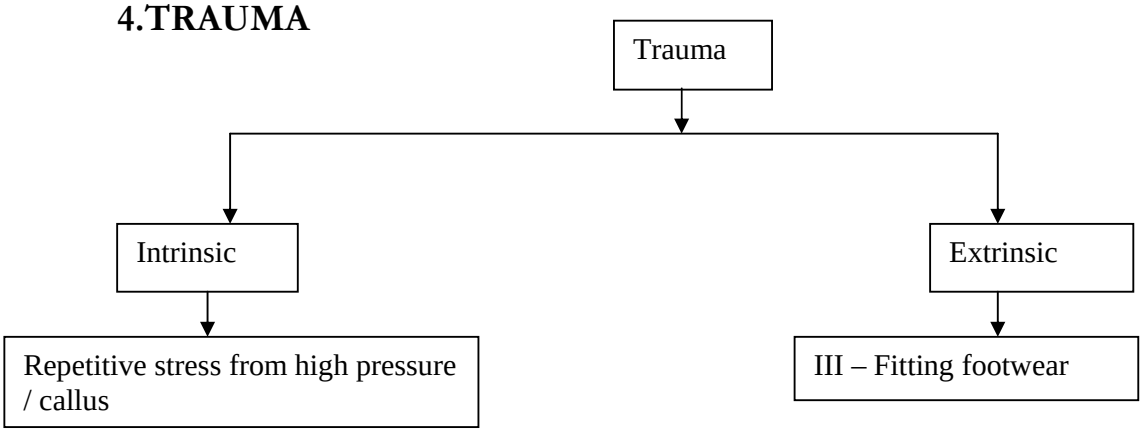
2. HIGH PRESSURE AT BONY PROMINENCES



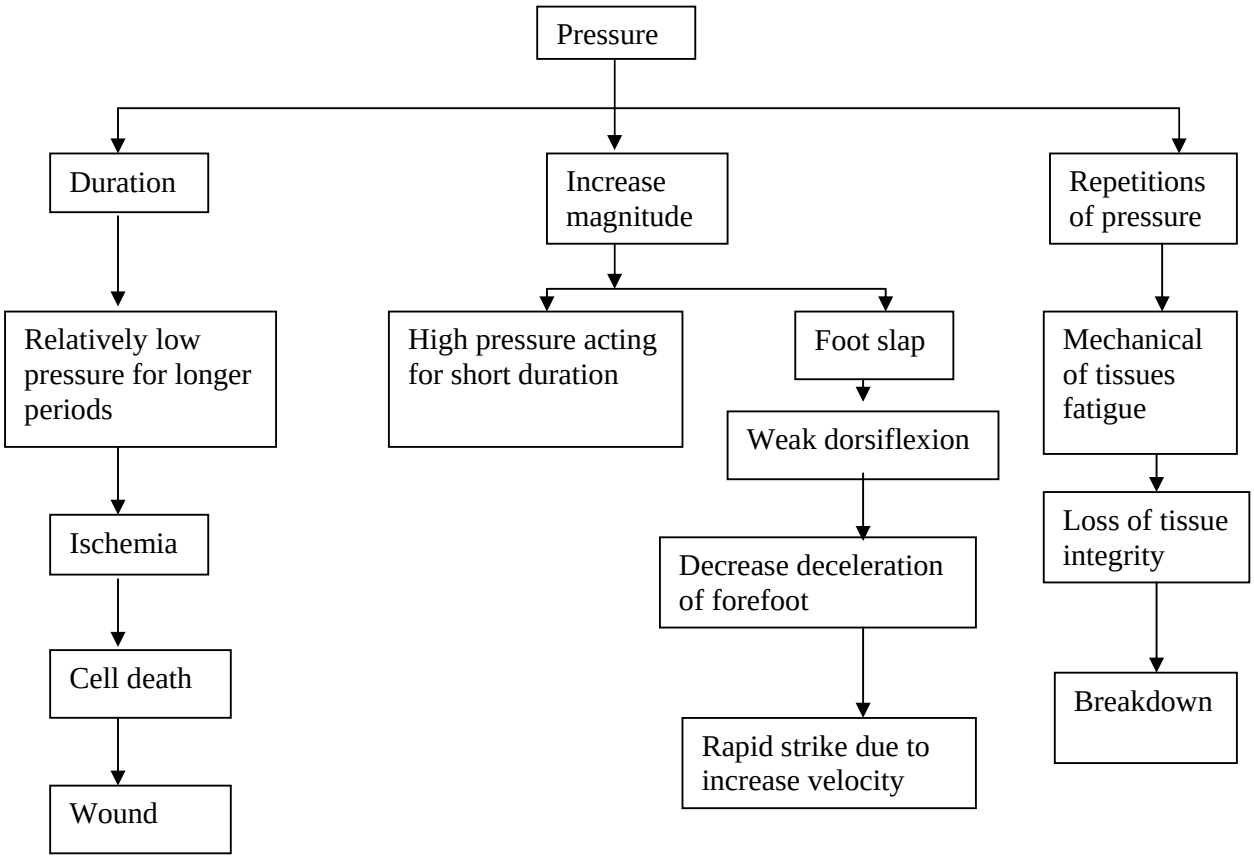
3. LIMITED JOINT MOBILITY



4.TRAUMA



Causation of ulceration



CLASSIFICATION OF DIABETIC FOOT

The Meggitt – Wagner classification is the most well – known and validated system for foot – ulcers

Grade	**Description**
Gade 0	**Pre or post – ulcerative lesion completely epithelialized**
Grade 1	**Superficial, full thickness ulcer limited to the dermis, not extending to the subcutis**
Grade 2	**Ulcer of the skin extending through the subcutis with exposed tendon or bone and without osteomyelitis or abscess formation**
Grade 3	**Deep ulcers with osteomyelitis or abscess formation.**
Grade 4	**Localized gangrene of the toes or the forefoot.**
Grade 5	**Foot with extensive gangrene**

ULCERS

Diabetic ulcers are classified clinically as

- **Neuropathic ulcers**
- **Ischemic ulcers**
- **Neuro ischemic ulcers**

NEUROPATHIC ULCERS

Develop at areas of high plantar pressures. (metatarsal heads, plantar aspect of the great toe, heel or over bony prominences in a charcot – type foot). Neuropathy is present in about 85-90% of foot ulcers in diabetic patients. Are painless, unless they are complicated by infection.

There is callus formation at the borders of the ulcer. Its base is red, with a healthy granular appearance. On examination evidence of peripheral neuropathy (hypoesthesia for complete loss of sensation of light touch, pain, temperature and vibration, absence of Achilles tendon reflexes, abnormal vibration perception threshold, often above 25v, atrophy of the small muscles of the feet, dry skin and distended dorsal foot veins) is present. However the pattern of sensory loss may vary considerably from patient to patient.

The foot has normal temperature of may be warm. Peripheral pulses are present and the ankle brachial pressure index in (n) or above 1.3

ISCHEMIC ULCERS

Ischemia is a major factor in 38-52% of cases of foot ulcers. These ulcers develop on the borders or the dorsal aspect of the feet and toes or between toes. They are usually painful.

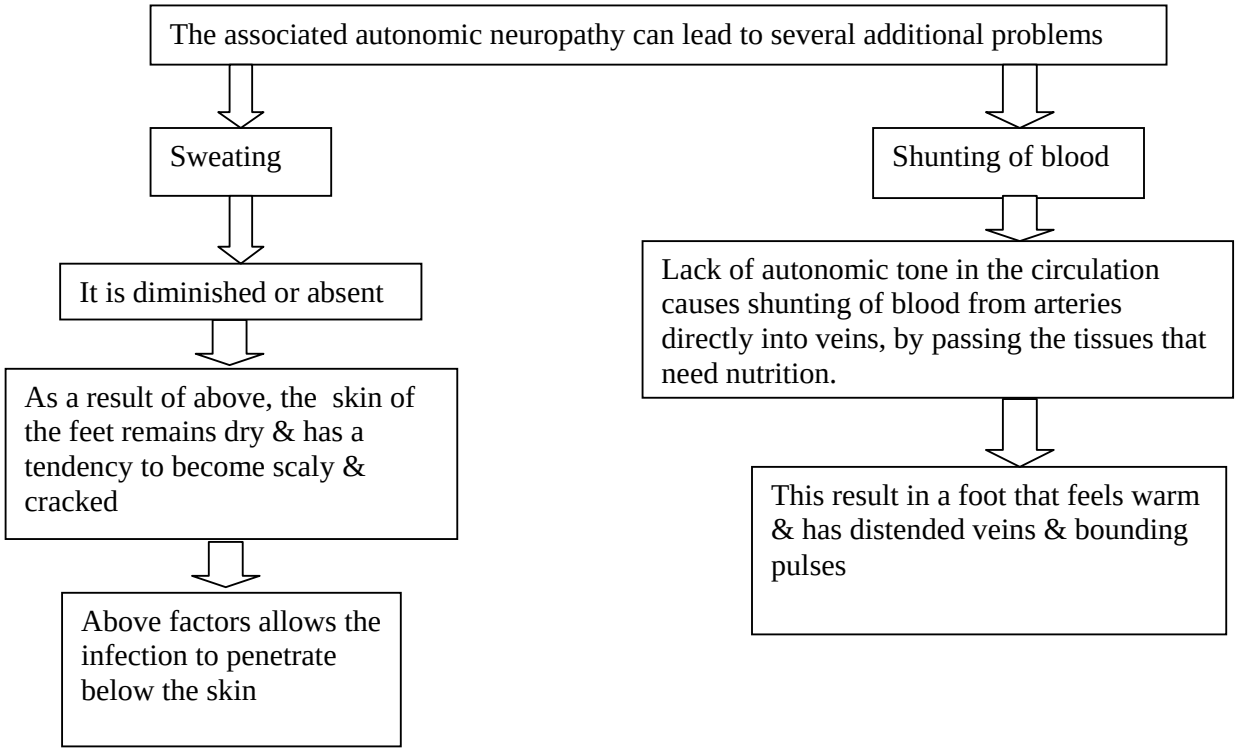
There is usually redness at the borders of the ulcer. Its base is yellowish or necrotic (black). There is history of intermittent claudication. On examination signs of peripheral vascular disease (skin is cool, pale or cyanosed, shiny and thin, with loss of hair and onychodystrophy; peripheral pulses are absent or weak, the ankle – brachial index is <0.9) are present.

Non – invasive vascular testing (duplex or triplex ultrasound examination, segmental pressures measurement, plethysmograpy) and angiography confirm peripheral vasculopathy.

NEURO –ISCHEMIC ULCERS (MIXED ETIOLOGY ULCERS)

Neuro ischemic ulcers have a mixed etiology ie.. neuropathy and ischemia

PATHOPHYSIOLOGY OF AUTONOMIC NEUROPATHY IN DIABETICS



Despite these apparent signs of adequate perfusion, the foot is vulnerable to local "Micro Vascular" gangrene, will heal very poorly and slowly, and will be less able to resist infection.

MANAGEMENT OF DIABETIC FOOT LESIONS

In 1999, the American Diabetes Association recognized several basic principles of diabetic wound healing.

- **Off – loading**
- **Debridement**
- **Use of appropriate dressings**
- **Medical and surgical treatment of infection**
- **Vascular reconstruction and / or amputation or reconstructive foot surgery when necessary.**

1. OFF LOADING OR PRESSURE RELIEF DEVICES

As has already been mentioned, biomechanical changes are a frequent consequence of diabetic neuropathy resulting in an altered pressure load on the sole of the foot. Therefore consistent pressure relief is an essential precondition for the prevention and healing of foot ulcers.

Total contact casting (TLC) is the most effective method of off – loading. A total contact cast in a special cast designed to redistribute the patient's weight off the ulcer site allowing ambulation while the ulcer is healing.

OFF LOADING TECHNIQUES.

Accommodative dressings	**-**	**patellar tendon – bearing braces**
Assistive devices	**-**	**removable walking braces**
Callus removal	**-**	**scotch cast boot**
Foot casts	**-**	**shoe cutouts**
Half, wedge or surgical shoes	**-**	**surgical correction of deformity**
Orthoses	**-**	**therapeutic shoes**
Padded hosiery	**-**	**total contact casting**

2.DEBRIDEMENT

Debridement of an ulcer is the corner stone of the management of active, acute or chronic wounds. The aim of debridement is to remove fibrin (white, yellow or green tissue seen on the bed of an ulcer) and necrotic tissue (black tissue) and to produce a clean, well vascularised wound bed.

Types of debridement are as follows:-

2.1SHARP SURGICAL (using scalpels) the gold standard for wound preparation, removes both necrotic tissue and micro – organisms. Majority of diabetics have neuropathy hence, fell no pain, therefore extensive sharp debridement or even operations on the feet can be performed without anesthesia.

2.2MECHANICAL using wet – to – dry dressings, hydrotherapy, wound irrigation and dextranomers.

2.3ENZYMATIC (using chemical enzymes such as collagenase, papain or try or trypsin in a cream or ointment base) – chronic wounds are enzymatically debrided in elderly patients when regular, sharp debridement is not possible, e.g.if the necrotic zone is thin, in ulcers with sinuses; and as an additional procedure to sharp debridement.

2.4AUTOLYTIC DEBRIDEMENT; using in vivo enzymes which self – digest devitalized tissue such as hydrocolloids, hydrogels, hydrogels, and transparent films. This uses the body's own enzyme and moisture to re – hydrate, soften and finally liquefy hard eschar and slough. It is selective, as only the necrotic tissue is liquefied and painless to the patient. Its main indication is non – infected ulcers with mild to moderate exudates.

2.5BIOMECHANICAL WOUND TREATMENT: (BIOSURGERY)

Treatment with sterile maggots (larval therapy).

The mechanism of action by which the larvae of Lucillia serricate (Greeenhottle fly) contribute to the cleaning to the cleaning and healing of necrotically coated or infected wounds has yet to be fully elucidated. The production of an antibiotic – like agent (also effective against micro – organisms resistant to conventional antibiotics). The presence of growth factors in the larval secretion, the destruction of bacteria by absorption and change in the ph value of the wound are postulate.

Leeches (Hirudo medicinalis) are also used in amputation surgery. Because of their local anti-inflammatory and antithrombotic effect due to the formation of hirudin, they are used in areas of critically impaired circulation or in the development of hematoma.

After debridement and infection control, the raw are is allowed to heal by (i) granulation, (ii) applying split skin graft or local random flaps or pedicled muscle flaps.

3. DRESSINGS: broad spectrum of wound dressing materials currently are available.

TABLE NO 3.5 :ADVANTAGES AND DISADVANTAGES OF AVAILABLE TYPES OF DRESSINGS

Type of dressing	Advantages	Disadvantages
Traditional dressings (gauze and absorbent cellulose)	Cheap and widely available appropriate for gangrenous lesions	Adhere to the wound bed and may cause bleeding o removal. Provide little protection against bacteria contamination
Films	Semi – permeable,. Form bacteria barrier. Durable, require changing every 4-5 days. Cheap	Useful on flat or superficial wounds only. Some patients are allergic to the adhesive in the dressing
Foams	Appropriate for ulcers with high production of exudates. Provide thermal insulation. Easily conformable, may be used to fill cavities without sinus tracts	Effect difficult to quantify. Not as effective and rapid as surgical debridement. Not appropriate for neuro – ischemic ulcers, which produce minimal exudates. Wound must be monitored closely for signs of infection
Hydrocolloids	Safe and selective, using the body's own defense mechanisms. Good for necrotic lesions, with light to moderate exudates. May be used to fill cavities without sinus tracts. Can be easily used with a shoe. Adhesive surface prevents slippages. Do not require daily dressing changes. Cost effective	Their occlusive and opaque nature prevents daily observation of the wound. Wound must be monitored closely for signs of infection. May promote anaerobic growth and mask a secondary infection.
Alginates	Useful as absorbents of exudates. Good for infected ulcers. Some products have hemostaic properties.	Not appropriate for neuro – ischemic ulcers, which produce minimal exudates. May dry out and form plug with in the wound bed. Requires painstaking removal with the use of large amounts of saline.
Enzymatic dressings.	Good for any wound with a large amount of necrotic debris, and for eschar formation. Promote autolysis and fast healing decrease maceration of the skin and risk of infection	Costly, must be applied carefully only to the necrotic tissue. May require a specific secondary dressing. Irritation and discomfort may occur.
Medicated dressings		Data based on animal models and cell cultures only.

4. SURGICAL MANAGEMENT OF DIABETIC FOOT

- **Surgical decompression of foot and leg**
- **Role of amputation**
- **Role of vascular management**

4.1 SURGICAL DECOMPRESSION – 3 types

FOREFOOT DECOMPRESSION

Web space infection, central plantar space infection are the indications. Incision should be placed deep into plantar space cutting plantar aponeurosis.

PLANTAR SPACE DECOMPRESSION

Main indication is a plantar space infection. Characteristic factor of this abscess is disappearance of longitudinal arch and skin crease. The area of longitudinal arch may bulge, sole is edematous, incision is made from little toe to heel over the medial aspect.

FOOT AND LEG DECOMPRESSION (fasciotomy)

Vertical incision for leg and horizontal for foot abscess, cellulitis are done.

AVERAGE HEALING TIME

Forefoot decompression	**-**	**11 – 38 days**
Plantar decompression	**-**	**12 – 40 days**
Foot and leg decompression	**-**	**12 – 60 days**

ROLE OF AMPUTATION

Factors deciding amputation are

1. **Age**
2. **Nephropathy**
3. **Major vessel disease**
4. **Gross neuropathy**
5. **Presence of gangrene**
6. **Involvement of bone**
7. **Uncontrolled diabetic ketoacidosis**
8. **Septicemia**

TYPES OF AMPUTATION

1. **Toe amputation**
2. **Great toe amputation**
3. **Other toes amputation**
4. **Ray amputation**
5. **Trans metatarsal amputation**
6. **Below knee amputation**

5. VASCULAR MANAGEMENT

1. **Role of pentoxyphylline**
2. **Antiplatelet durgs**
3. **Surgery – Endarterectomy / Bypass Procedure**

6. ADJUVANT THERAPY FOR WOUND HEALING

1. **Cultured human dermis and cultivated equivalents**
2. **Hyperbaric oxygen therapy**
3. **Ketanserin**
4. **Growth factors**
5. **Granulocyte – colony stimulating factor**
6. **Electrical stimulation**
7. **Sulodexide**
8. **Hyaff**
9. **Low level laser therapy**

RECOMMENDATIONS.

- **The feet should be examined at least annually in patients with type 2 diabetes & in those with type – 1 diabetes for more than 5 years.**

- **A detailed neurological examination & assessment for peripheral vascular disease should be performed**

- **We recommend using the quantitative foot assessment for neurological symptoms & sings described above, including the 5.07 U mono-filament test**

- **Patients should be considered at particularly high risk for future plantar ulceration if they have**
- **A previous H/o foot ulceration or amputation**
- **Neuropathic foot deformities, especially with overlying bunions or calluses,**

PROPHYLACTIC FOOT CARE

It is important that prophylactic advice on foot care be given to any patient whose feet are at high risk. The recommendations for prophylactic foot care are –

Avoid

1. **Smoking**
2. **Walking barefoot**
3. **The use of heating pads or hot water bottles**
4. **Stepping into a bath without checking the temperature**

The feet should be:

- **Washed daily in tepid water**
- **Mild soap should be used and the feet should be dried by gentle patting**
- **A moisturizing cream or lotion should then be applied.**

Toe Nails:

- **Trimmed to the shape of the toe**
- **Filed to remove sharp edges.**

Shoes

- **The patient's shoes should be snug, not tight,**
- **Patients who have misshapen feet or have had a previous foot ulcer may benefit from the use of special customized shoes.**

- **A prospective study found that shoe variables other than the recommendation for customized shoes (e.g. style, width, length or type of shoe) had no preventive effect.**

The use of customized shoes, however, reduced the development of new foot ulcers from 58 to 28 percent over one year of follow – up in a second report

Socks:

- **Cotton**
- **Loose fitting**
- **Should be changed every day**

Inspection of feet:

The feet should be inspected daily, looking between and underneath the toes and at pressure areas for skin breaks, blisters, swelling, or redness. The patient may need to use a mirror or if vision is impaired, have someone else perform the examination.

Examination of foot by medical person:

A particularly effective strategy is to make specific recommendations to the patient in the form of a "contract'. And to advise the patient to request that his or her feet be examined at every visit to the doctor or nurse.

IDEAL DIABETIC FOOT TEAM

Diabetic foot requires a team work to save the foot. The team consists of:

- **General surgeon**
- **Dietician**
- **Podiatrist**
- **General physician**
- **Microbiologist**
- **Pharmacist**
- **Nurse**
- **Orthopaedician**
- **Vascular surgeon**
- **Plastic surgeon**
- **Physiotherapist**
- **Laboratory specialist (foot Pressures)**

HYPOTHESIS

Is low level laser therapy given daily with clustered probe at 2.5 m W at 5KHz frequency, 10,600 nm wavelength (CO_2 Laser) for 2 - 5 min depending on wound area at a dose of 8 - 10 J/cm2 along with conventional treatment compared to conventional therapy alone, effective in reducing the mean percentage in area reduction significantly in patients with diabetic foot ulcers more than 3 weeks old admitted in C M C H under surgery department.

MATERIALS AND METHOD

STUDY DESIGN-RANDOMIZED CLINICAL TRIAL:

The present study was carried out at Coimbatore medical college hospital for a period of two years. 40 patients with diabetic foot ulcers participated in present study, using protested and pre- designed Performa based on which the study was randomized into either group using a computerized randomization chart. Out of 40 partcipants,20took treatment in form of conventional therapy and the remaining 20 had treatment with low level laser therapy(LLLT) along with conventional therapy.

SAMPLE SIZE:

Total of 40 patients of which 20 patients in control group received conventional therapy and study group received low level laser therapy along with conventional therapy (in form of betadine and saline dressings and wound debridements).

PERIOD OF STUDY

A randomized control study from October 2007 to September 2009.

INCLUSION CRITERIA:

- *Type 2 diabetes mellitus*
- *Diabetics between 40 and 70yrs of age*
- *Size of ulcer more than 8X8X1.5cm*
- *Duration of ulcer more than three weeks duration*
- *Grade I diabetic wound Wagner's classification*
- *Negative wound cultures*

EXCLUSION CRITERIA:

- *Pulse less limb*
- *Immunocompromised patients*
- *Hemoglobin level 10mg*
- *Associated osteomyelitics*
- *Cardiac connective disorder*
- *Cellulitis*
- *Diabetic ketoacidosis*
- *Diabetic gangrene*
- *Connective tissue disorder*
- *Uncontrolled hypertension and renal failure*
- *Surgical interventions done during part 15 days*
- *Pregnant women*
- *Infected ulcers*
- *Patients who discontinued treatment while under study due to migration or intolerance to laser therapy also were excluded.*

After participants enrolled to study, informed consent taken from patient, after explaining the procedure. Patients randomized with the help of a computer generated randomization chart. Participants of study group received laser therapy along with conventional treatment, while those in control group received only conventional mode of treatment.

RECORDING OF DATA:

Detailed history and through clinical examination done in all cases. Documentation done using a stratified Performa which included demographic data of patients, details of investigations, treatment provided and measurement of wound areas of patients enrolled in study.

INVESTIGATION:

- *Hematological,biochemical,microbiological and radiological investigation carried out as enumerated in Performa using standard procedures.*
- *Urine analysis, fasting blood sugar, renal parameters, culture of wound and x-ray of local part done as out patient procedure.*
- *Peripheral pulses examined manually and also confirmed with hand held Doppler.*
- *Wound area measured by tracing the margin of ulcer in a transparent film and counting the number of squares on a graph underneath.*

PROCEDURE:

Initial wound measurement taken in both the groups before staring their respective treatment, that is conventional treatment in control group and conventional treatment along with LLLT in study group.

TREATMENT OF STUDY GROUP

- **PART PREPARATION**: *Saline irrigation given to entire wound area to clean the wound before laser treatment and allowed to air dry.*

- *Carbon di oxide (co2) laser having wave length of 10,600 A units used in the study*

- *Laser irradiation given s per protocol given below.*

TABLE NO 5.1 : LASER IRRADIATION PROTOCOL

Power	*Duration*	*Energy Density*	*No.of Days*	
2 mW	*4 sec /cm2*	*8J / cm2*	*Day 01*	*Scanning dose*
2.5 mW	*4 sec / cm2*	*10 J / cm2*	*Day 04,07,10,13,16,19*	*Over entire wound area*

- *On day 01, scanning dose given at a dose of 8J/cm2, tolerability, reactions or any side effects to laser therapy in patients noted.*

- *If patients tolerates scanning dose with no adverse effects, treatment schedule is continued once in 3 days, i.e. on day 04,07,10,13,16,19 with a dose of 10J/cm2.*

- *After treatment, an adhesive sterile bandage applied over the treated wound. Subjects instructed to remove bandages only to shower, reapply new bandages after the shower and replace bandages if removed.*

- *Conventional treatment in form of daily moist dressing using saline, betadine, wound debridement given as and when required.*

- *All subjects compiled with treatment for entire 20 days period.*

SAFETY MEASURES:

- *Both subject and the administrator wore laser safety goggles before, during and after treatment to prevent damage to retina in study group.*

- *All investigators wore latex gloves during procedures.*

CONTROL GROUP:

- *All patients instructed to both daily in morning and wash wound with plain soap & water wound and allow it to air dry.*

- *conventional treatment in form of moist dressing using saline, antibiotic treatment, wound debridment given daily depending on wound.*

- *After treatment, sterile dressing given and patients instructed to remove it only next day for shower.*

- *Al patients compiled with treatment for 20 days.*

- *All investigators wore later gloves during procedure.*

STERILIZATION:

All instruments and dressing materials sterilized before use, by standard methods.

ASSESSMENT OF WOUND:

- *Subjects from both groups returned on 20th day for assessment of wound and measuring of wound.*

- *Efficiency of low-level laser therapy (LLLT) assessed by measuring wound area on day 01 and day 20th in both control & study group and subjected to statistical analysis.*

OBSERVATION AND RESULTS

TABLE NO 6.1 : SEX DISTRIBUTION

CASES

Sex	No.of.Cases	Percentage
Male	12	60
Female	8	40
Total	20	100

CONTROLS

Sex	No.of.Cases	Percentage
Male	13	65
Female	7	35
Total	20	100

BOTH

Sex	No.of.Cases	Percentage
Male	25	62.5
Female	15	37.5
Total	40	100

In our study it was observed diabetic foot occurs more commonly in males (62.5%) as compared to females (37.5%)

TABLE NO 6.2 : AGE OF DISTRIBUTION

CASES

Age	No.of.Cases	Percentage
41-50	4	20
51-60	11	55
> 60	5	25
Total	20	100

CONTROL

Age	No.of.Cases	Percentage
41-50	5	25
51-60	12	60
> 60	3	15
Total	20	100

BOTH

Age	No.of.Cases	Percentage
41-50	9	25.5
51-60	23	57.5
> 60	8	20.0
Total	40	100

In our study, it was observed diabetic foot was commonest in the age group between 51-60 yrs of age (57.5%)

TABLE NO 6.3 : MEAN AGE OF GROUPS

STUDY GROUP	Age of Population Percentage	
	Mean	SD
Cases	55.24	4.64
Controls	52.84	6.24

Mean age groups in cases and controls were 55.24 and 52.84 respectively which were statistically not significant

Mean age of both the groups is 54.04

TABLE NO 6.4 : SOCIO ECONOMIC LEVEL OF PATIENTS STUDIED

Socio – Economic Class	NO. of Patients	Percentage
Poor Socio Economic Status (Monthly Income <Rs.1,000)	30	75.0
Lower Middle Class (Monthly Income : Rs.1,000 – Rs. 6,000)	7	17.5
Upper Middle Class (Monthly Income > Rs 6,000)	3	7.5
Total	40	100

Greater prevalence of Diabetic foot ulcer was observed in poor socio – economic class since most of them were manual laborers prone to trauma and due to poor hygiene

TABLE NO 6.5 : TYPE OF FOOT WEAR AMONG PATIENTS INSTUDY

Foot Wear	Total No. of Cases	Percentage
Walk Bare Foot	15	37.5
Wear Slippers or Chapels	20	50
Appropriate foot wear	5	12.5
Total	40	100%

IN our study, about 87.5% of patient wear no appropriate foot wear.

TABLE NO 6.6 : DURATION OF DIABETES CASES

Duration in (yrs)	Total no. cases	Percentage
1 – 5	12	60
6 – 10	6	30
11 - 15	2	10
Total	20	100

CONTROL

Duration in (yrs)	Total no. cases	Percentage
1 – 5	11	55
6 – 10	7	35
11 – 15	2	10
Total	20	100

BOTH

Duration in (yrs)	Total no. cases	Percentage
1 – 5	23	57.5
6 – 10	13	32.5
11 – 15	4	10.0
Total	40	100.0

In our study it was observed that 57.5% foot ulcers has occurred in patients who has diabetes mellitus for durations of one to five years.

TABLE NO 6.7 : ANTI DIABETIC AGENTS

CASES

Anti - Diabetics	No. of Cases	Percentage
OHA	14	70
Insulin	6	30
Total	20	100

CONTROL

Anti - Diabetics	No. of Cases	Percentage
OHA	13	65
Insulin	7	35
Total	20	100

BOTH

Anti - Diabetics	No. of Cases	Percentage
OHA	27	67.5
Insulin	13	32.5
Total	40	100

In our study, majority of participants were on OHA (67.5%) compared to patients taking insulin (32.5%).

TABLE NO 6.8 : FASTING BLOOD SUGAR

CASES

FBS (mgs)	No. of Cases	Percentage
80-110mg	6	30
111-125mg	8	40
126-150 mg	6	30
Total	20	100

CONTROL

FBS (mgs)	No.of Cases	Percentage
80-110mg	7	35
111-125mg	8	40
126-150 mg	5	25
Total	20	100

BOTH

FBS (Mgs)	No.of Cases	Percentage
80-110mg	13	32.5
111-125mg	16	40
126-150 mg	11	27.5
Total	40	100

In our study almost (72.5%) had fasting blood sugar values less than 125mg%. Only 27.5% had values more than 126mg%.

TABLE NO 6.9 : MODE OF DIABETIC FOOT ULCERS

CASES

Type of onset	No .of patients	Percentage
Traumatic	12	60
Spontaneous	8	40
Total	20	100

CONTROL

Type of onset	No.of patients	Percentage
Traumatic	13	65
Spontaneous	7	35
Total	20	100

BOTH

Type of onset	No.of patients	Percentage
Traumatic	25	62.5
Spontaneous	15	37.5
Total	40	100

In our study, Trauma is most common cause of origin of diabetic foot ulcer (62.5%) while only (37.5%) are spontaneous in origin.

TABLE NO 6.10 : SITE OF ULCER

CASES

Site	No.of patients	Percentage
Plantar	13	65
Dorsum	7	35
Total	20	100

CONTROL

Site	No.of patients	Percentage
Plantar	11	55
Dorsum	9	45
Total	20	100

BOTH

Site	No.of patients	Percentage
Plantar	24	60
Dorsum	16	40
Total	40	100

In our study, Diabetic ulcer foot more commonly occurs on plantar aspect (60%) of foot as compared to dorsal aspect (40%)

TABLE NO 6.11 : WOUND CONTRACTION

Group	Mean reduction %	S.D	Median	'P" value
Control	15.55	2.58	11.3	P<0.001
Study	41.17	3.15	37.67	P<0.001

In our study, it was observed that mean percentage of area reduction was higher in study group (41.15) as compared to controls (15.5)

TABLE NO 6.12 : WOUND CONTRACTION RELATED TO SITE

Group	Plantar			Dorsal		
	No	Mean reduction %	S.D	No.	Mean % Reduction	S.D
Control	11	14.66%	1.72	9	16.27%	2.20
Study	13	38.45%	2.11	7	41.86%	2.34

In our study, it was observed patients with ulcers over plantar aspect has lesser percentage of mean wound area reduction as compared to participants with wound over dorsal aspect, in both control group and study group.

STATISTICAL ANALYSIS

- ***Statistical analysis was done by using computer programs and standard mathematical formula.***

- ***Diabetic foot ulcers in the study group had better mean percentage of wound contraction of 41.15 percentage (S.D; 3.15 : median ; 37.67) as compared to the control group which had had mean percentage of wound contraction of 15.55 percentage (S.D; 2.58, Median; 11.3) the difference in the mean percentage of area reduction of the two groups when studied using unpaired student T test was found to be significant ($p < 0.001$).***

DISCUSSION

Every surgeon's desire is that after dressing the wound, it should heal without any complications. Successful wound dressing should keep the wound moist and devoid of any adverse reactions such as infection, maceration and allergy. Diabetic ulcer are chronic wound, stuck in inflammation phase and shows cessation of epidermal growth. It is projected that developing countries will experience the greatest risk in the prevalence of type-2 diabetes in next twenty years.

Low laser therapy (LLT) has shown great promise as a procedure for healing of chronic wounds.

The present study was conducted at Coimbatore Medical College Hospital, Coimbatore to study the effect of Low level Carbon-di-oxide (Co_2) laser therapy on chronic diabetic wound healing dynamics.

1. **In present study it was seen that the incidence of diabetic foot ulcers were more in males 62.5 % (25 patients) as compared to females 37.5 % (15 patients). The second national data source, NHDS documented higher hospital rates in males suffering from diabetic foot ulcer.**

2. **Diabetic foot ulcers are most common in 6^{th} decade 57.5 % (23 patients), while only 24.5 % (9 patients) affected in 5^{th} decade. Older patients have more chances of developing diabetic foot ulcer. The prevalence of diagnosed diabetes increase with age. The NHDS, study undertaken in USA between 2002 and 2004 also revealed that elderly diabetics has twice risk of developing a foot ulcer.**

3. **Mean age of both study and control group is 54.04 years. Age group involved in our study is similar to that reported from Karl Franzens University, Austria where mean age is 56.2 years.**

4. **In our study, about 75 % (30 patients) of participants belonged to poor socio-economic status with a monthly income of less than Rs. 1,000. Most of them are manual laborers who are more prone to trauma, poor hygiene, lack of balanced diet are adding to increased incidence of diabetic ulcer. The confounding factor is that in Government hospital set-up people from high socio-economic status do not prefer to get treated. Hence, the exact comparison of prevalence among various socio-economic classes was not feasible in this study.**

5. **As most participants are from poor socio-economic status due to illiteracy, poverty 87.5 % (35 patients) of our participants do not wear appropriate foot wear. Either they are bare foot walkers or wear inappropriate sized foot wear. A study by Edmonds et al 1986 shows most of diabetic foot ulcers are invariably shoe related and due to gait abnormalities. They can be prevented by appropriate sized foot wear and educating to avoid tight foot wear.**

6. **In our study, it was observed 57.5 % foot ulcers has occurred in patients who has diabetes mellitus for one to five years, followed by participants having diabetes 6-10 years, constituting 32.5 %**

7. **Most of patients 67.5 % (27 patients) were on oral hypo-glycemic drugs for control of sugar whereas only 32.5 % (13 patients) were on insulin.**

8. **In our study almost 72.5% (29 patients) had FBS values less than 125mg% and only 27.5% had [11 patients] values more than 126mgs% this suggests that participants in both groups had a good glycemic control overall.**

9. **In this study 62.5 % of ulcers (25 patients) were traumatic in origin, trauma being the triggering factor secondary to neuropathy. About 37.5 % (15 patients) were spontaneous in origin secondary to blister rupture or unnoticed trivial trauma.**

10. **Around 60 % (24 patients) of participants had ulcer on plantar surface of fore foot and remaining 40 % (16 patients) on dorsum of foot. Edmonds et al showed 68 % of foot ulcers were on plantar and fore-foot areas. As already mentioned inappropriate foot-wear is a major reason.**

11. **It was also observed in our study that patients who had ulcers on plantar aspect of foot in control group had mean wound contraction of 14.66% [S.D.=1.74] as compared to dorsal wounds of same group which had mean contraction of 16.27% [S.D=2.25]. Similarly the mean plantar wound contraction in the study group was 38.45% [S.D=2.13] as compared to mean dorsal wound contraction 41.86% [S.D = 2.36] suggesting different wound healing dynamics in two regions of foot.**

12. **In our study, participants receiving Low Level Laser Therapy(LLLT) along with conventional therapy had better wound contraction 41.77% [S.D = 3.15] , as compared to the group receiving only conventional treatments in whom the mean wound contraction was 15.55%. [S.D = 2.58], these were found to be statistically significant on unpaired student 'T' test ($p < 0.001$), suggesting LLLT enhances wound healing in diabetic wounds.**

FEASIBILITY OF THIS STUDY

1. **In a study [low level laser therapy facilitates superficial wound healing in humans, a triple – blind sham – controlled study] performed by I Ty Hopkins, Todd A.Mcleod, Jeff G. Seegmiller , G. David Baxter published in journal of athletic training 2004; 39(3) 223-229 repotted results in 22 healthy subjects and shown 55% greater wound contraction of cases as compared to controls**

2. **In present study we have taken 40 patients from type – 2 diabetes mellitus with ulcers. Patients were taken up for study based on inclusion and exclusion criteria. Out of 40 patient, 20 patient [12 male, 8 female] were cases and 20 patient [13 males, 7 females] were. control. Participants included in the study group were irradiated with 2mw, 8 J/cm2 for 2-5 min on day 01 [scanning dose] and then2.5 mw, 10J/cm2 for 2-5min over entire wound surface for once in 3 days in 20 days periods, accounting for seven LLT sessions. The wound area is radiated at prescribed power[2mw or 2.5mw] for 4 sec /cm2. On calculating, depending on wound area LLLT is given for a duration of 2-5min . The dose delivered is 8-10J/cm2**

   (Joules = Power X Duration of exposure)

3. **All 40 patient from both study and control group compiled strictly to the 20 days treatment schedule.**

4. **The initial area measurement for both groups taken on day 01 and final area measurement on day 21 taken on transparency sheet and number of squares counted on a graph paper underneath.**

5. **We applied the following formula to calculate percentage reduction in area of wound after 20 days period in period in both cases and control groups**

$$\text{Rate of contraction of wound} = \frac{(\text{initial area} - \text{final area})}{\text{Initial area}} \times 100$$

6. **We have found 15.55% (S.D. = 2.58) contraction of wounds in control groups as compared to 41.77% (S.D. = 3.15) contraction of wounds in study group. Therefore, study groups are having 25.62% more wound contraction as compared to control group. On applying, unpaired student T test ($p<0.001$) which is significant.**

7. **In a study [low level laser therapy facilitates superficial wound healing in humans, a triple – blind sham – controlled study] performed by I Ty Hopkins, Todd A.Mcleod, Jeff G. See miller , G. David Baxter published in journal of athletic training 2004; 39(3) 223-229 repotted results in 22 healthy subjects and shown 55% greater wound contraction of cases as compared to controls**

8. **But in our study we have found only 25.62% greater wound contraction for cases as compared to controls. The difference in results between our study and mentioned above may be due to the fact that all patients in above study were non – diabetics, otherwise healthy humans with no co – morbid illness. Whereas, patients in our study were suffering with diabetes mellitus type – 2 and its associated complications.**

From our study we can say that Low – Level CO2 Laser therapy facilitates wound healing in patient suffering from diabetes mellitus type – 2 along with surgical dressings.

SIDE EFFECTS

- **Many patient complained increase in serous discharge from wound during laser therapy.**
- **Very minority of patients had intractable serous discharge and itching due to laser therapy, compelling them to discontinue LLLT.**
- **Some patients had mild burning pain lasting for 24 hrs on the day of laser session.**
- **The laser therapist had episodes of headache, nausea, irritability, straining of eyes during some prolonged laser sessions.**

LIMITATIONS OF OUR STUDY

- **Not a blinded study**
- **Follow up is short to derive conclusion on long term healing of ulcers**
- **Cost involved not analyzed in this study.**
- **LLLT can be used only for ulcers of moderate size (<15 cm2) to produce optimum results**

RECOMMENDATIONS:

- **Large scale clinical trials involving large no of participants should be done to prove efficacy of LLLT beyond doubt.**

- **Detailed study of LLLT and its hazardous effects is necessary to protect patient and therapist from laser hazards.**

SCOPE FOR FURTHER STUDY:

There is further scope of study is with low level laser therapy in diabetic wounds with respect to anti – infective properties of low level laser therapy as shown by recent studies by Andrei p.sommer et al (Anti – infective and low level light. A new chapter in photo medicine and laser surgery 2007; 25(3) :150 – 158)

SUMMARY

The incidence of diabetes and complications are on rise. Diabetic foot being one of the most common complication. 15% of all diabetics develop diabetic ulcers, the most common site being the foot. Diabetes has highest risk factor associated with limb threatening ischemia. Trivial trauma secondary to neuropathy and distorted pedal architecture causes ulcerations. . 20% of admission in diabetics are for foot problems.

Various modalities of treatment have been developed to aid faster healing of diabetic foot ulcers. Course of healing in diabetic foot patients is unpredictable and resistant to treatment.

- **40 patients of diabetic foot ulcers were studied. They were divided into two groups of 20 each.**
- **One group received Low Level Laser therapy along with conventional therapy and the control group received treatment in the form of conventional therapy. A comparative study was done between both groups regarding percentage of wound area reduction.**
- **All patients in the study underwent X-ray of the affected foot, patients with stress fractures and osteomyelitis were excluded.**
- **23 patients were between 51-60 years of age. Males were more affected than females. 62.5% males vs 37.5% females.**
- **Mean age of both the groups is 54.04**
- **87.5% of patient wear no appropriate foot wear, whom needed education regarding foot wear.**

- **57.5% foot ulcers has occurred in patients who has diabetes mellitus for durations of one to five years.**

- **Majority of participants were on OHA (67.5%) compared to patients taking insulin (32.5%)**

- **Almost (72.5%) had fsting blood sugar values less than 125mg% only 27.5% had values more than 126mg%**

- **Trauma is most common cause of origin of diabetic foot ulcer (62.5%) while only (37.5%) are spontaneous in origin.**

- **Diabetic ulcer foot more commonly occurs on plantar aspect (60%) of foot as compared to dorsal aspect (40%)**

- **It was observed patients with ulcers over plantar aspect has lesser percentage of mean wound area reduction as compared to participants with wound over dorsal aspect, in both control group and study group.**

- **In our study it was observed that participants receiving LLLT along with conventional treatment had better wound contraction of 41.15% as compared to the group receiving only conventional treatment in whom the mean wound contraction was 15.55% these were found to be statistically significant on unpaired student T test ($p<0.001$) enhances wound healing in diabetic wounds.**

Thus, low level laser therapy in the treatment of diabetic foot ulcers was found to be more effective, safe, promoter of wound healing, and hence can be recommended for the treatment of diabetic foot ulcer as an adjuvant to the conventional mode of treatment.

CONCLUSION

- **The wounds in subjects treated with LLLT contracted more than the wounds in the non treated group 41.15% vs 15.55% p = <0.001 significant) . This indicates LLLT as an effective modality to facilitate wound contraction in patients suffering from diabetes and can be used as an adjunct to conventional mode of treatment (dressings and debridment) for healing of diabetic wounds.**

- **Surgical laser is safe for patient and operating personnel, relatively sample to use, rapid in its action and without untoward impact on tissue locally or at a distance.**

- **The mechanisms effectively responsible for cell mitotic activity have not been clarified yet.**

- **Therefore, we noted that there is a need for research on the action and parameters of low-intensity laser .Clinical trials with higher sample size are proposed to evaluate the efficacy of low level therapy in treatment of this type of wounds.**

- **Progression of foot ulcer from one grade to another can be prevented, thus avoiding involvement of bone and its complications and save the limb.**

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PHOTOGRAPHS

CO2 MEDICAL LASER

LASER THERAPY BEING ADMINISTERED

SAFETY GOGGLE

STUDY GROUP

(CO_2 LOW LEVEL LASER THERAPY)

DAY 1 **DAY 20**

DAY 1 **DAY 20**

STUDY GROUP

(CO_2 LOW LEVEL LASER THERAPY)

DAY 1

DAY 20

DAY 1

DAY 20

CONTROLGROUP

(CONVENTIONAL THERAPY)

DAY 1

DAY 20

DAY 1

DAY 20

RESEARCH PARTICIPANT INFORMATION AND CONSENT FORM

Mr./ Miss/ Mrs._____

You are invited to participate in our research study, "EFFICACY OF LOW LEVEL LASER THERAPY IN ULCER FOOT HEALING IN TYPE – 2 DIABETIC PATENTS "

Since you are suffering from diabetes and foot ulcer, which is not healing since a long time and will be requiring treatment for the same, you are eligible to be a part of the study and hence asked to participate. This research is about the beneficial effects of Low Level Laser Therapy on your foot ulcer and the result of this research will help in a better treatment of similar participants in the future. If you agree to be a part of this research, we would ask you some relevant clinical history. You are free to not to answer to whichever question you think are not relevant. A clinical examination will be done and then you will be given Low Level Laser Therapy along with the regular betadine dressings for once in three days. On the first day the area of the ulcer will be measured and this will be repeated at the end of the therapy that is 21st day.

There are chances you may have a speedy and better recovery with this therapy and it will also help in the treatment of participants with similar complaints in the future. Your decision of whether or not to participate in this study will not affect the quality of treatment you receive. Further you may withdraw from the study at any time. All the new information collected about you during the course of this study will be kept

confidential to the extent permitted by the law. Any information, which identifies you personally, will not be released without your written consent.

This study doses not have any damaging aspect and there are no chances of injury during the course of the study, but if injured the investigator is not responsible. There will be no extra cost incurred by you. The participation in this study is entirely voluntary and you may withdraw from the study at any time. At any time during or after the study, for any information you may contact the researcher.

Dr.R.Senthil Kumar

Room No: 53, PG & CRRI Men's Quarters

Coimabatore Medical College Hospital

Coimbatore – 641 018.

Ph : 98404 04305

Signature of the participant or legally authorized representative:

Participant's Name :

Signature :

Experimenter / Witness's Name :

Signature :

Date :

Place :

PROFORMA

PATIENT IDENTIFICATION DATA:

Name **IP/OPD NO.**

Age **DOA**

Sex **DOD**

Address

CHIEF COMPLAINTS:

MEDICAL HISTORY:

- **Peripheral Neuropathy ( )**
- **Nephropathy ( )**
- **Retinopathy ( )**
- **PVD ( )**

DIABETIC STATUS:

- **Type :**
- **Duration :**
- **Medication : Oral Hypoglycemics Insulin**
  ( ) ( )

ULCER DETAIL:

- **Mode of onset Traumatic ( )**
  Spontaneous ( )

- **Duration**
- **Progress**

WOUND OBSERVATION:

- **Site**
- **Size**
- **Shape**
- **Edge**
- **Margin**
- **Floor**
- **Base**
- **Discharge**
- **Surrounding skin**
- **Contractor**

NERUROLOGICAL EXAMINATION

VASCULAR EXAMINATION	**Left**	**Right**
Popliteal a.	**( )**	**( )**
Ant.Tibial	**( )**	**( )**
Post Tibial	**( )**	**( )**
Dorsalis Pedis	**( )**	**( )**

ANY FOOT DEFORMITY PRESENT

- **Toe deformity**
- **Bunion**
- **Charcots foot**
- **Foot Drop**

IF AMPUTATION HAS BEEN DONE

Specify **Date :**

Side :

Level :

Cause for amputation :

FOOT WEAR ASSESSMENT: Does Patient wear appropriate shoes

INVESTIGATION

- **CBC**
- **FBS 1st** _____ **Date:** _____

  2nd (24 hr apart) _____ **Date:** _____

- **Sr.Creatinine**
- **Urea**
- **Urine :**

  Routine

  Microscopy

- **X-ray Foot**

  AP View

  Lateral View

- **Wound C/s**

WOUND AREA MEASUREMETN ON D 1 IN CM2

LLT SPECIFICATIONS

Duration :

Power :

Frequency :

Dose :

Once in Three Days :

ANALYSIS PLAN

TOTAL CALCULATED DOSE OF LLT IN JOULE _____

Date	Day	LLT
	1	1
	4	2
	7	3
	10	4
	13	5
	16	6
	19	7

Ulcer area Measurement

Day 1 ☐ **cm^2**

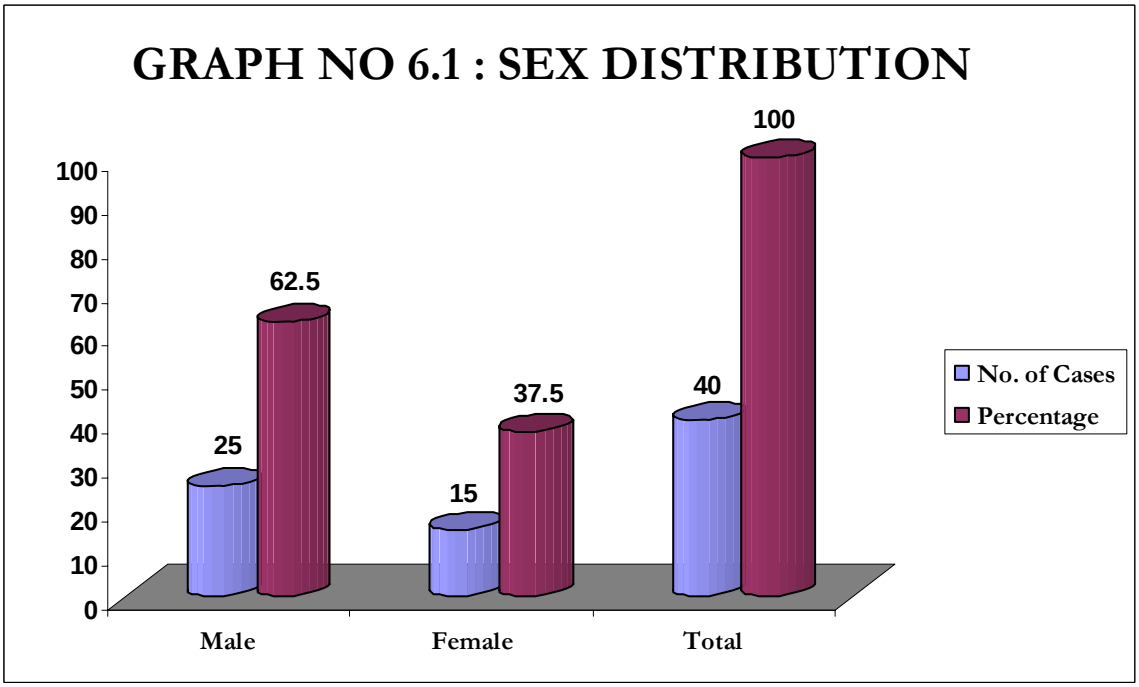
Day 20 ☐ **cm^2**

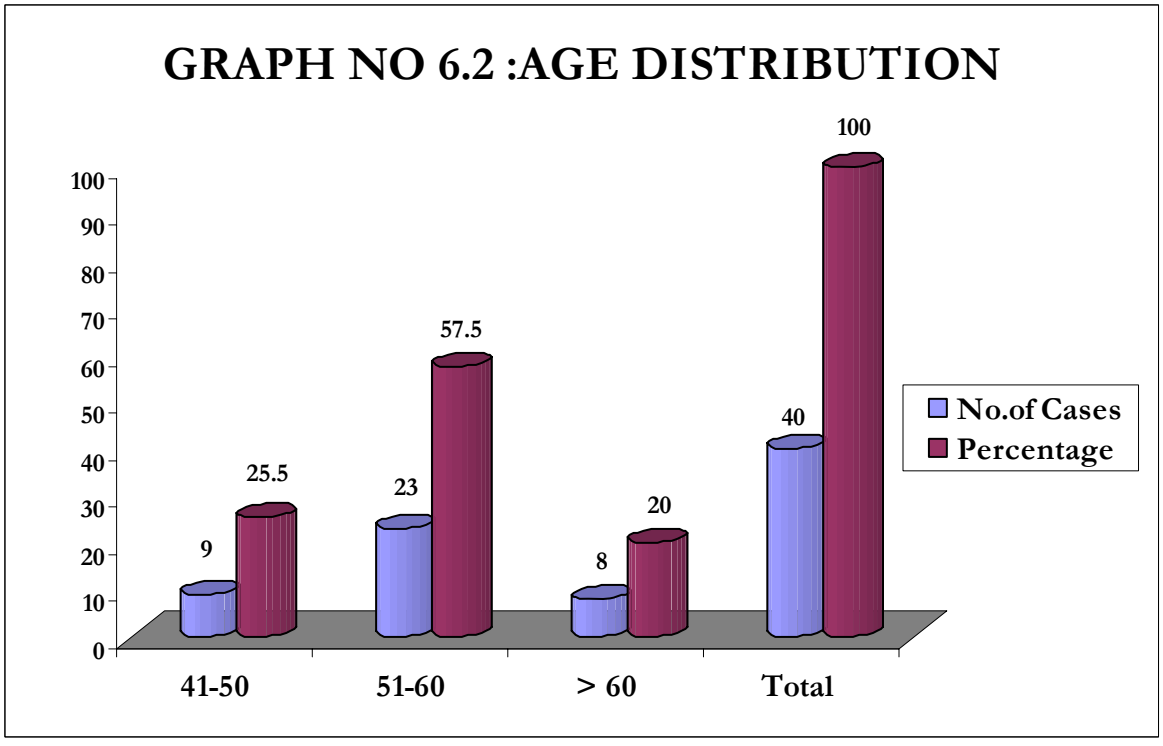
$$\text{\% Area Reduction} = \left(\frac{\text{Initial Area} - \text{Final Area}}{\text{Initial Area}} \right) \times 100$$

KEY TO MASTER CHART

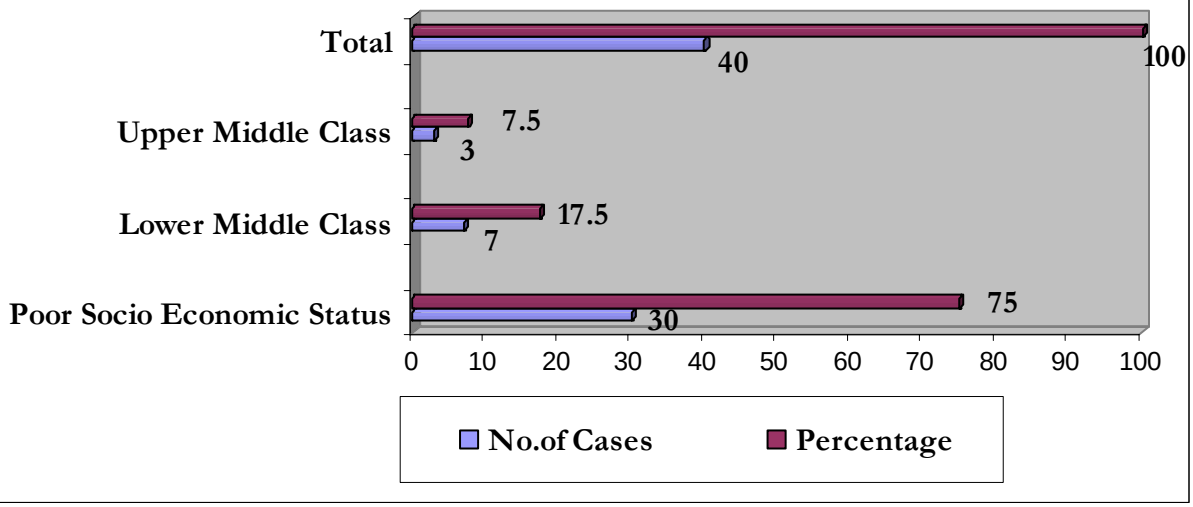
SL.No	Serial Number
IP No	In Patient Number
S	Spontaneous
T	Traumatic
M	Male
F	Female
I	Insulin
O	Oral Hypoglycemic
DM	Diabetes Mellitus
Rx	Treatment
FBS	Fasting Blood Sugar
N	Normal
NOGC	No Organisms Grown
IA	Initial Area
FA	Final Area
CA	Calculated Area
P	Plantar
D	Dorsal
C/S	Culture Sensitivity

GRAPHICAL REPRESENTATION

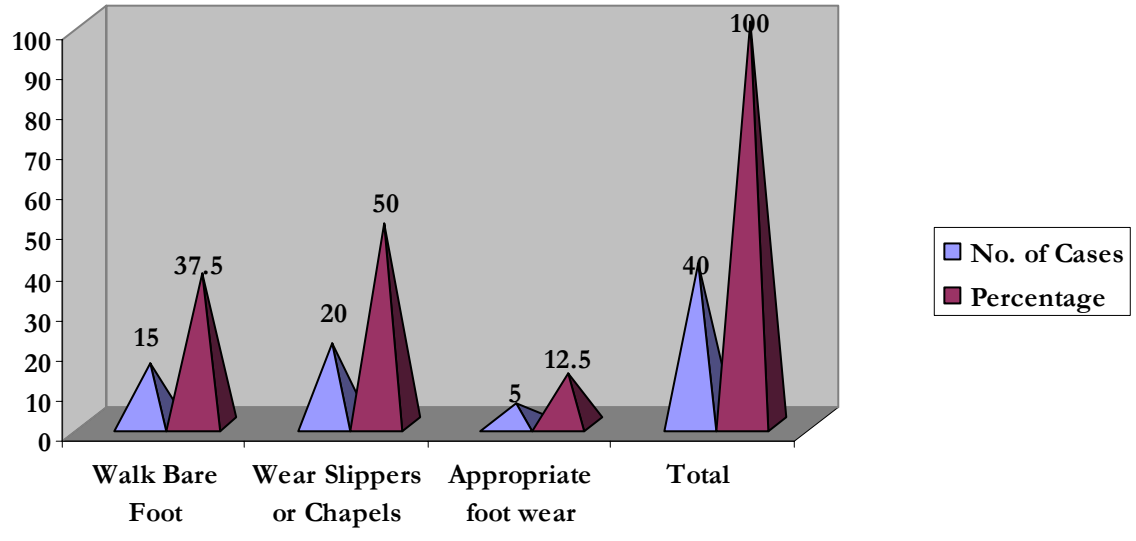


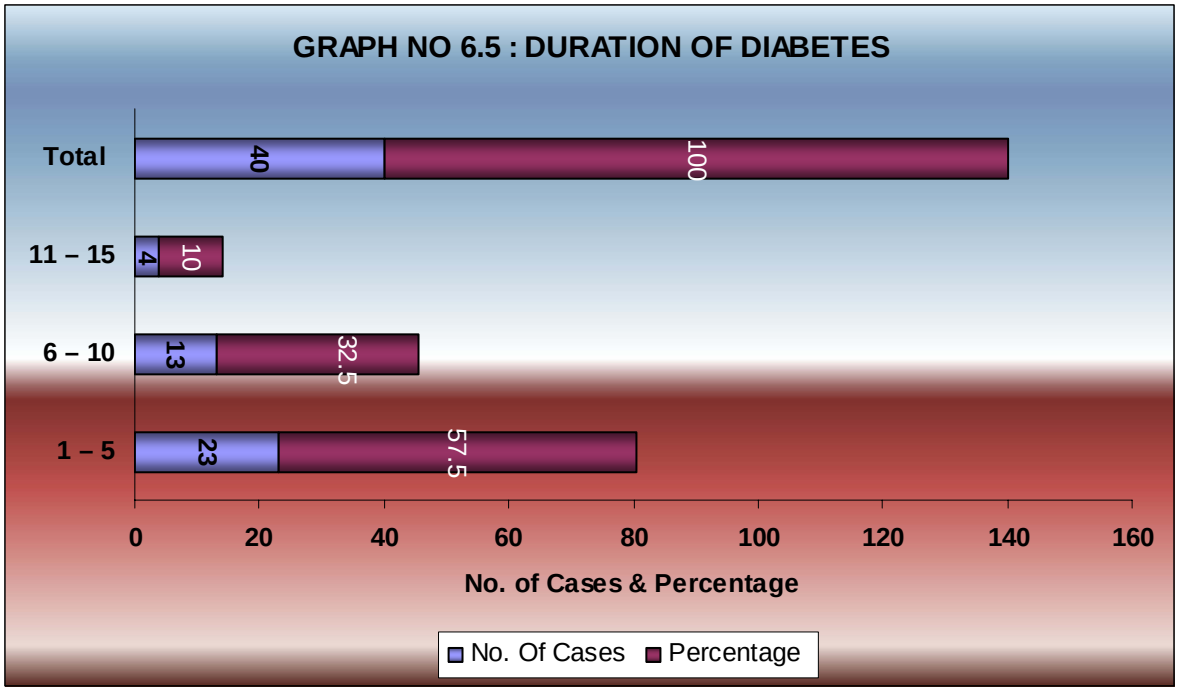


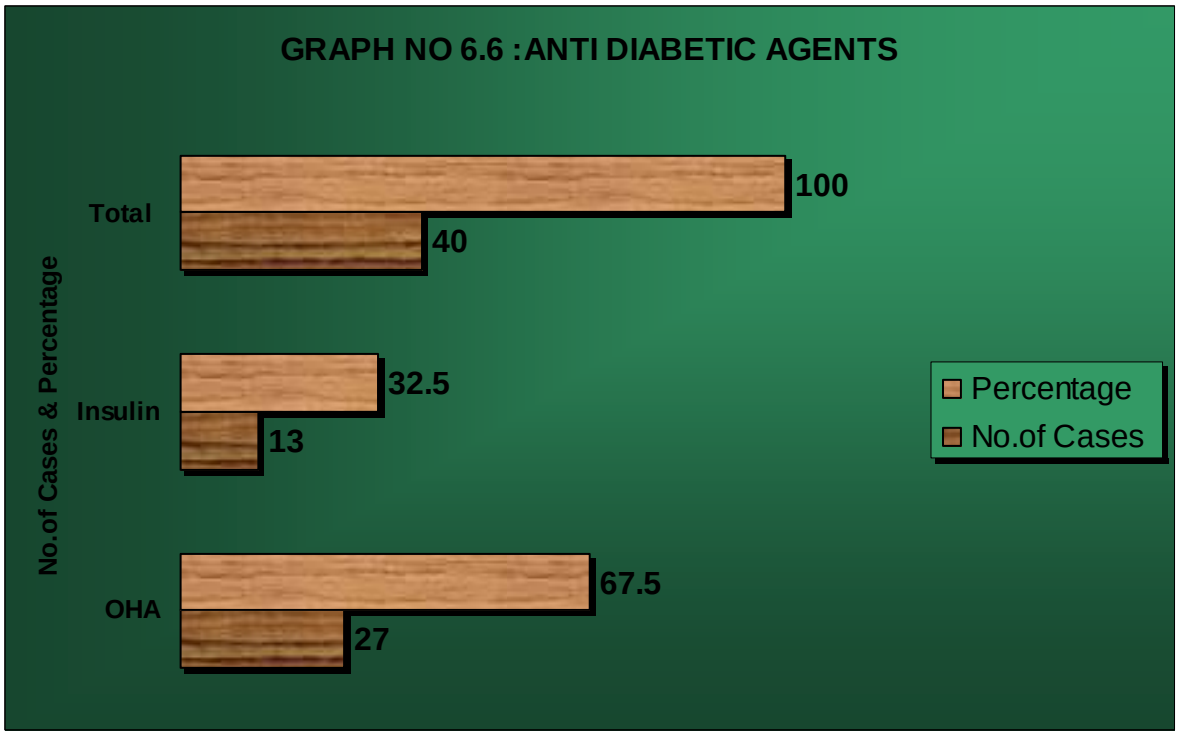
GRAPH NO 6.3 : SOCIO ECONOMIC LEVEL OF PATIENTS STUDIED



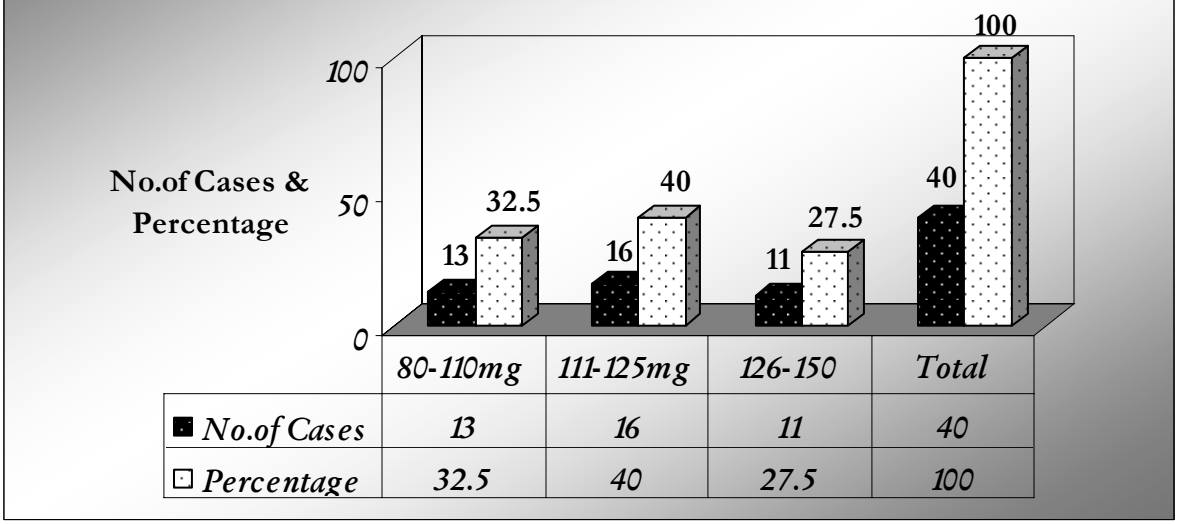
GRAPH NO 6.4 :TYPE OF FOOT WEAR




GRAPH NO 6.5 : DURATION OF DIABETES
Total
11 – 15
6 – 10
1 – 5
40
100
4
10
13
32.5
23
57.5
0
20
40
60
80
100
120
140
160
No. of Cases & Percentage
No. Of Cases
Percentage

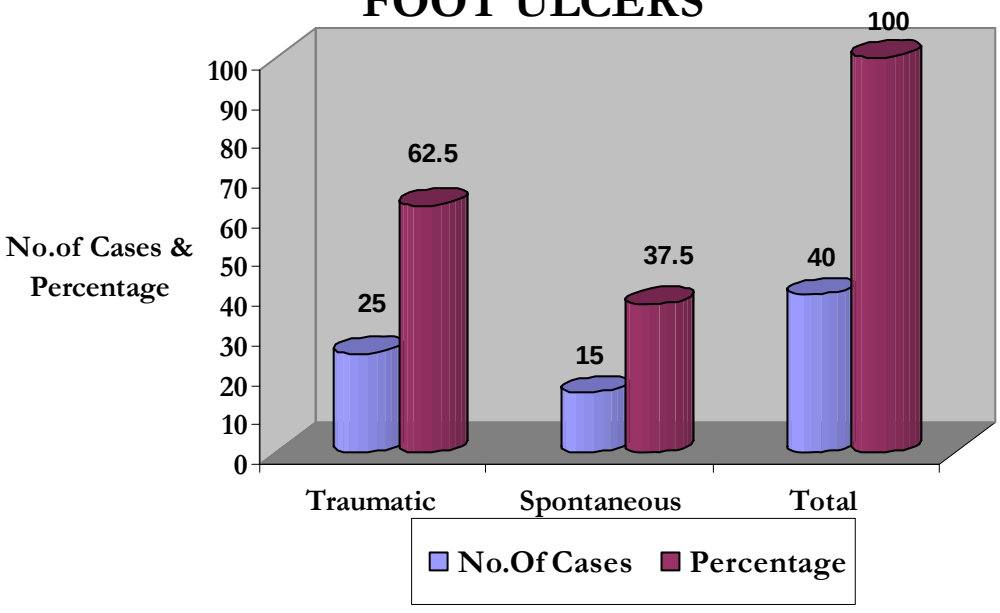

GRAPH NO 6.6 :ANTI DIABETIC AGENTS
No.of Cases & Percentage
Total
Insulin
OHA
100
40
32.5
13
67.5
27
Percentage
No.of Cases

GRAPH NO 6.7 :FASTING BLOOD SUGAR

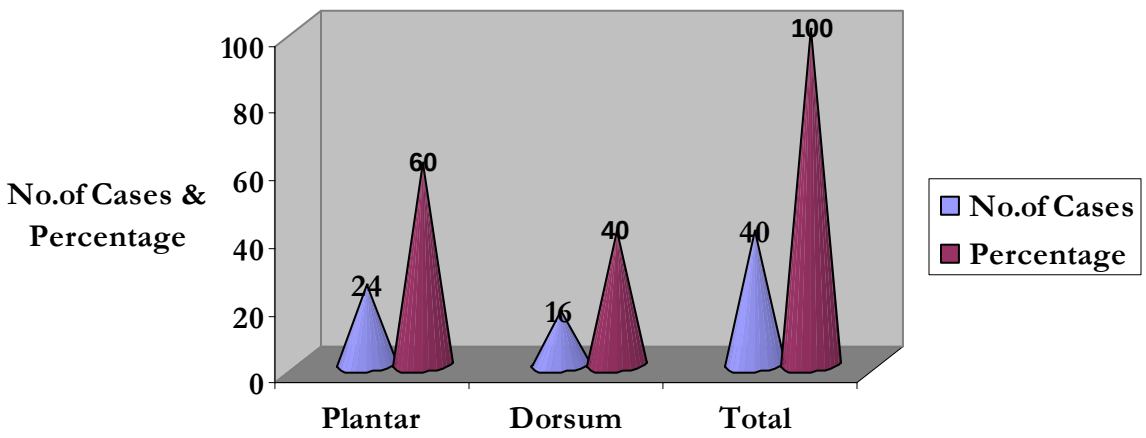


	80-110mg	111-125mg	126-150	Total
No.of Cases	13	16	11	40
Percentage	32.5	40	27.5	100

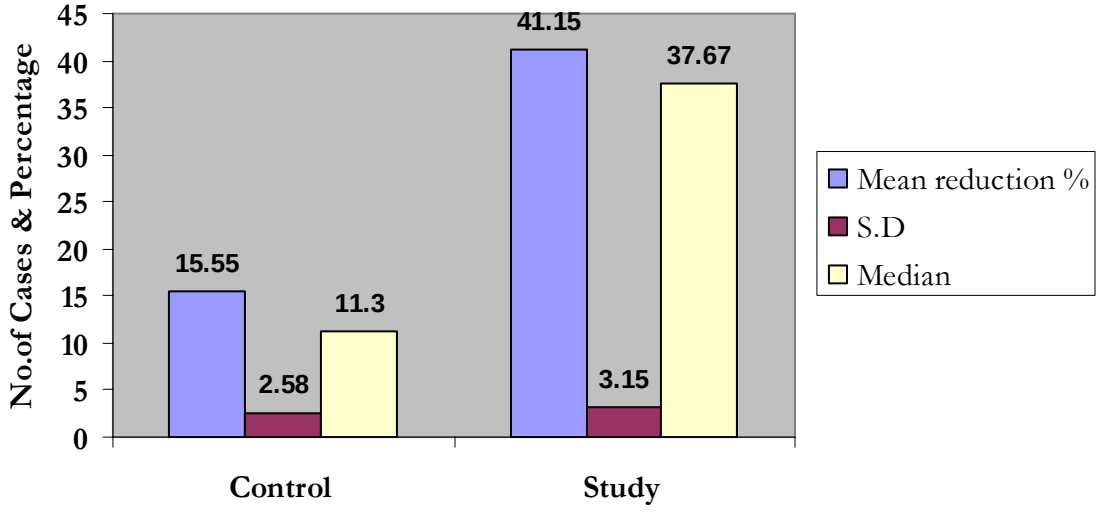
GRAPH NO 6.8 : MODE OF DIABETIC FOOT ULCERS



GRAPH NO 6.9 : SITE OF ULCER



GRAPH NO 6.10 :WOUND CONTRACTION



MASTER CHART CONTROL GROUP

SI.No	IP No O.P.No	Age / Sex	Onset	Site	Anti DMR	TBS	X-Ray	C/S	Initial Area MM2	Final area mm2	Ia –fa = ca	% area reduction
1.	41120	50/M	T	D	O	100	N	NOGC	250	200	50	20%
2	42465	55/M	S	P	I	135	N	NOGC	540	430	110	21.2%
3	47202	58/M	S	D	O	136	N	NOGC	1450	1200	250	17.3%
4	48315	53/M	T	P	I	150	N	NOGC	550	420	130	18.2%
5	49363	58/F	S	P	O	118	N	NOGC	450	360	90	12%
6	50493	45/M	S	D	O	145	N	NOGC	2150	1850	300	13%
7	50586	76/M	T	P	O	86	N	NOGC	1400	1150	250	17.4%
8	50798	52/M	T	P	O	120	N	NOGC	1950	1650	300	15%
9	51898	44/M	T	P	I	108	N	NOGC	1050	910	140	14.5%
10	52765	48/F	T	D	O	124	N	NOGC	450	380	70	19.6%
11	53421	44/M	T	P	I	140	N	NOGC	350	300	50	14.2%
12	54921	52/M	S	P	O	94	N	NOGC	300	250	50	18%
13	55655	54/M	T	D	O	122	N	NOGC	450	410	40	8.8%
14	57863	55/M	T	D	O	90	N	NOGC	610	490	120	16.5%
15	58963	62/F	S	P	O	96	N	NOGC	560	490	70	12.2%
16	59215	48/F	T	D	I	116	N	NOGC	905	800	105	11.3%
17	60812	53/M	S	D	O	104	N	NOGC	1150	1050	100	13.2%
18	61151	56/F	T	P	O	112	N	NOGC	290	240	50	18%
19	62851	57/F	T	P	I	115	N	NOGC	460	380	80	15.2%
20	63573	65/F	T	D	I	120	N	NOGC	520	400	120	17%

MASTER CHART STUDY GROUP

SI.No	IP No O.P.No	Age / Sex	Onset	Site	Anti DMR	TBS	X-Ray	C/S	Initial Area mm^2	Final area mm^2	IA –FA = CA	% area reduction
1.	40120	73/F	T	P	O	120	N	NOGC	2050	1550	500	36.8%
2	41363	52/F	S	D	O	116	N	NOGC	540	320	220	40.4%
3	40202	55/M	T	P	I	100	N	NOGC	580	360	220	41.7%
4	41311	58/M	S	P	O	96	N	NOGC	555	365	190	34.2%
5	41361	42/M	T	P	O	130	N	NOGC	1550	950	400	38.3%
6	41493	58/F	S	D	O	115	N	NOGC	110	25	85	77%
7	41580	70/F	T	P	O	136	N	NOGC	775	325	450	42%
8	42586	60/M	S	P	I	90	N	NOGC	150	25	125	80%
9	42597	60/M	T	P	O	138	N	NOGC	525	350	175	38.3%
10	42683	62/M	S	D	O	105	N	NOGC	250	125	125	50%
11	42700	69/M	T	P	I	70	N	NOGC	175	50	125	71%
12	48335	58/F	T	P	O	125	N	NOGC	725	550	175	39.2%
13	49150	48/M	T	P	O	118	N	NOGC	875	525	350	40.0%
14	51199	52/M	S	D	I	124	N	NOGC	540	320	220	36.2%
15	55379	49/M	T	D	O	128	N	NOGC	1250	750	500	40%
16	55365	45/M	T	D	O	14	N	NOGC	500	250	250	50%
17	59335	65/F	S	P	I	88	N	NOGC	250	160	90	42.4%
18	59395	44/F	T	P	O	112	N	NOGC	1300	690	610	46.8%
19	1685	51/F	S	P	O	118	N	NOGC	650	400	250	38.4%
20	1790	53/M	T	D	I	142	N	NOGC	850	525	325	38.2%